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United States
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Agriculture

Forest Service

Pacific
Northwest
Region

October 1987



Managing Competing and Unwanted Vegetation

Draft Environmental Impact Statement

Appendices



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Appendix A

Timber Growth and Yield Analysis

A

Appendix A

Timber Growth and Yield Analysis

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Introduction

There are about eight million acres (approximately one-third of all lands managed by the Pacific Northwest Region) that will be managed for "full timber yields" under the Forest Land Management Plans. These lands are expected to also yield high quality water, provide habitat and forage, and retain their potential capacity for production of both these tangible commodities and not-so-tangible values, such as rare species and plant diversity.

Even with an intensive management strategy, it is unlikely that maximum yield (the full biological site potential) and the elimination of all competition will be achieved in the management of these public lands. While competing vegetation is often detrimental to conifer growth, certain situations or growth stages may be beneficial. These potential benefits, noted by Walstad and Kuch (1987), include:

1. prevention of soil erosion on disturbed or unstable sites though protective canopies or root systems;
2. nutrient uptake, storage, and recycling that may otherwise be lost following harvest or other site disturbance;
3. improvement in soil physical and chemical properties through addition of organic matter and nutrients;
4. amelioration of excessively hot, dry, or cold (frost) conditions in new plantations through shading or mulching effects;
5. protection of seedlings from browsing or grazing animals, such as deer, elk, and cattle;
6. reduction or elimination of damage from conifer diseases (e.g., root rots) through effects on soil-borne pathogens.

A major problem is the lack of linkage between short-term observed effects, (such as growth response from reduced competition; growth reduction associated with nitrogen loss; and reduction in growth associated with the reduction of mycorrhizal populations) and other long-term yield effects. Research in these areas is not available. Long-term studies and record keeping are critical needs.

Regeneration is an ongoing process in nature, particularly after a disturbance such as fire or timber harvest. The competition for newly available space is fierce. There are usually an excess of individuals competing for the site's limited resources. Competition is so intense that usually many more individuals die than survive. Death soon after germination often results from lack of resistance to short-term climatic extremes. Thus, the total number of individuals sharply decreases, leaving the better adapted, more competitive plants. This process is

well known as “survival of the fittest.”

As individual plants grow, they require more space and resources, and natural thinning from direct competition results. There are numerous articles describing the effects of competition. White and Harper (1973) illustrate the “natural thinning curve” with farm crops, and Drew and Frewelling (1979) used 313 forest sites analyzing volume growth and tree density following the “ $-3/2$ power law” which gives the general form for the reduction of individuals (density) with an increase in volume production. (See Fig. No. 1). This principle is the basis for response from vegetation management activities. In a resource limited system (most are), as the numbers of individuals are reduced, the remaining plants will access the released space and resources.

Although the complex competitive interaction between and within species is not completely understood, the type and degree of response is known to depend on a number of site-specific variables. The species of crop tree, the species of the competition, their age, size, and vigor influence the amount of response. The intensity of competition by a species also depends on whether that species is near the optimum or limits of its environmental range. Variation in response associated with a species can be significant. With over 50 possible competitors and their varied response throughout the Region, much basic information is needed.

Water, nutrients, and light are the resources most often limiting. All affect growth and survival, but each has a different physiological effect. For example, height growth is achieved in a short period early in the growing season, but diameter growth can occur throughout. This means that early water shortages could affect both height and diameter growth, but later shortages may only affect diameter growth.

It is clear that a reduction in numbers of individuals or cover of the competition will result in a response from the crop tree. The general form of the response is shown in Figure 2, but the absolute values on the axis are dependent on site-specific conditions. These environmental variables and limiting factors must be carefully identified in the silvicultural diagnosis.

Assumptions and Qualifications

1. Cost-of-doing business and economic efficiency are not limitations. The reality of budget levels and economic thresholds are addressed in Chapters III (Affected Environment) and IV (Environmental Consequences) of the parent EIS document. This assumption is used to focus on environmental factors related to treatment effectiveness. Likewise,

the relative safety risks of techniques will be disregarded in this analysis, but are addressed elsewhere in the document.

2. Broadcast burning is assumed to be available if considered appropriate for meeting site preparation objectives. An analysis of the role of burning in the reforestation program is made in Chapter IV (Environmental Consequences).

3. Substitutability of site preparation or plantation release methods is assumed. In other words, there are no constraints due to project logistics or scale of operations. In practice, treatment options at the District or Forest implementation level can be severely restricted due to these factors. Realistic operational constraints, such as availability or terrain limitations, will be applied.

4. A generic strategy will be assumed for this analysis: even-aged management using artificial regeneration, intermediate thinnings where appropriate, and final harvest at the physical rotation age, (95 percent of culmination of mean annual increment of growth measured in cubic volume). This is done for ease of modelling timber yields over time, and to reflect a sequence of events often used on intensively managed timberlands in the Pacific Northwest Region. In actual practice, the selection of silvicultural systems for site-specific prescriptions are based on management objectives; limitations within the operational environment; vegetative response; and guidelines provided in Forest Land Management Plans or Regional Guides.

This assumption forces an extreme simplification of the diverse harvest systems, environmental conditions, and silvicultural strategies now being employed in the Pacific Northwest Region. Rotation length is a key variable in the estimation of timber yield effects. Short rotations, for example, will magnify the relative effects of early growth suppression in managed stands. Those used in this assessment will be the most representative management regimes for the Forest planning "full yield" timberland component of each specific combination of vegetation being analyzed.

5. Vegetation control is assumed to be geared primarily toward conifer seedling establishment and growth. In reality, these activities often have dual-purpose objectives. Examples are the controlled livestock grazing in established plantations to improve forage utilization as well as conifer release; burning for reduction of fuel accumulations and creation of favorable seedling microsites; or grass-sedge control in high elevation units in order to limit pocket gopher populations and subsequent tree damage.

6. Timely reforestation treatments following site disturbance (timber harvest or wildfire) has been assumed. This means that the status of conifer seedlings is initially approximately codominant or better with associated vegetation. Reforesting while brush-grass-

herbaceous invasion or development is still at manageable levels improves the probability of successfully meeting prescription objectives, and also keeps treatment options open. This implies that a degree of site preparation has occurred prior to or during reforestation efforts. The quality and intensity of site preparation can eliminate or delay the need for subsequent vegetation control.

Most of the available literature dealing with the effects of competition reflects studies conducted on sites where some disturbance of pre-existing vegetation has occurred. For this reason, the yield effects which would occur under Alternative C (no vegetation management) will be somewhat greater than those reflected in this assessment.

Unfortunately, the sheer size of the land base under management—and a budget-planning process that involves speculation over a several-year period—can occasionally result in an unacceptable time lag before treatment. When considered appropriate, this factor will be incorporated into the analysis. A typical operational problem is coordination of nursery sowing schedules with anticipated future timber harvest priorities. Several variables in the sequence of events needed to successfully reforest a site will influence the need for conifer release. This may include things such as seed availability, seedling quality, planting quality, or the success of a prescribed site preparation broadcast burning.

7. A moderate level of damage to crop trees during vegetation management is anticipated and considered acceptable in operational projects. Elements such as scattered foliar or leader damage during herbicide application; seedling trampling or browsing by livestock; or physical crushing or deformity caused by manually treating brush can occasionally be a severe site-specific impact.

Examples of unacceptable herbicide damage are documented by both Newton (1978), and by Gratkowski and Lauterbach (1974); while heavy damage during manual cutting is seen in Hobbs and Wearstler (1985), and Roberts (1980). In the great majority of treatments, however, crop tree damage is either transient in nature, or the tree loss not severe enough to cause a falldown in volume yields in the merchantable stands. Many of the studies being used in this analysis, in fact, record an inconsistent height increment response following treatment. This is partly a reflection of this individual crop tree damage during treatment. On a programmatic basis, a practice that consistently results in high levels of tree loss or deformity will simply be discontinued or modified to correct the problem.

8. The presence or absence of significant competition for conifer seedlings is directly related to the extent and volume of associated vegetation. Managed yield tables developed for Forest Plans are an

expression of conifer growth potential under a light to moderate degree of interspecies competition in newly established stands. Total elimination of associated vegetation is rarely the objective during plantation establishment and maintenance. This is a reflection of the trade-offs between the beneficial and negative aspects of woody shrubs and herbaceous vegetation. It also reflects the fact that total vegetation control is neither silviculturally feasible or necessary for attainment of management objectives.

A competition threshold level effect has been displayed in work by Oliver (1985), and others. An effort has been made to select studies used for growth comparisons which reflect a severity or degree of competition similar to that commonly found in operational prescriptions.

It is difficult, if not impossible, to extrapolate from highly site-specific growth effects to broad scale generalizations. Controlled experiments produce specific results that may not be representative of those occurring in a more heterogeneous forest environment. For this reason, there is an inherent danger of exaggerating broad scale timber yield effects based on controlled research. A degree of caution and conservatism is, therefore, incorporated into this analysis. Results from long-term monitoring studies eventually will provide answers to questions addressed in this EIS.

An analysis of this sort involves limitations:

1. There is a growing body of data and sources of information related to the effects of competing vegetation. All published information pertinent to Oregon and Washington, however, documents only a short time frame during the stand management cycle. This means that translation of early growth and yield effects into managed stand yields (over rotations that are typically 80 to 120 years) involves much extrapolation and uncertainty. There is also a general shortage of empirical information regarding long-term effects of vegetation management on the site productivity of western United States forest lands.

This lack of long-term stand monitoring presents a problem in displaying the reduction of tree growth and vigor which may linger in unreleased trees which grow through the competition.

A comparatively long-term study (28 years) reported in Strothmann and Roy (1981) indicates that tree vigor differences (expressed as needle complement, needle length, and needle color) continue to be apparent in released and unreleased trees. Most data used in this analysis covers a period of less than 10 years. In order to account for this "memory" of growth suppression and loss of vigor (due to reduced crown or root system development, form defect, rooting depth, etc.), the short-term height and diameter increment trends will be

projected to age 15 to 20. At this point, the unreleased conifer is assumed to have established sufficient dominance over competing vegetation in order to grow at rates comparable to a tree which has been "free-to-grow" throughout its life. In other words, the growth curve is simply interrupted for the first 1-1/2 to 2 decades. There is, however, some evidence that understory brush can cause serious growth suppression in older sapling and poletimber stands (Oliver 1984, and Barrett 1982).

During the second decade of stand development, intraspecific competition may become more significant than interspecific effects. On full-yield or intensively managed sites, however, stocking control and density management principles are generally employed during young stand tending.

2. The literature dealing with the effects of competing vegetation on conifer growth is occasionally erratic or conflicting. An effort was made to review the available information. The experience and opinions of USDA Forest Service field practitioners were used in those cases where the literature is inconclusive. For consistency with the NEPA implementing regulations, only published information and data (or that on file and reasonably available to interested parties) will be utilized.

3. Potential yield effects due to seedling or sapling mortality are measured against the stocking levels used to certify plantations as successful. These are tree numbers which provide the commercial thinning yields anticipated in many of the managed stand projections used in Forest Plan development.

4. This assessment is made within limitations of time and data availability, and is intended as an estimate of consequences within NEPA guidelines. Several long-term comprehensive studies are currently underway that will help identify treatment alternatives, growth and yield effects, and cost factors. The high priority nature of these efforts and the need for carefully designed study protocol are illustrated in Owston et al. (1986).

Examples are an effort begun in 1980 in northern California through the Pacific Southwest Forest and Range Experiment Station, the Pacific Northwest Station's Treatment Monitoring Study, and a study in southwestern Oregon started in 1983 through the Forest Intensified Research (FIR) Program of Oregon State University. Some individual Forests are also conducting administrative reviews or studies to monitor and use vegetative growth effects information. An example is a Siskiyou National Forest interpretation of on-Forest monitoring plots. These results will not be available for this EIS. However, as more precise or reliable information such as this becomes available, it will be utilized in project planning and implementation at

the Forest level.

5. There is uncertainty involved in extrapolation from site-specific results to general observations, based on a limited number of studies. This uncertainty is then compounded with the expansion by acreage estimates. For this reason, the timber yield effect estimates for each vegetation complex will involve some large statistical error terms. These estimates, however, are adequate for a display of the magnitude of change between alternatives in the EIS. It should be emphasized, however, that these estimates simply represent the approximate mean of a range of results.

Yield Reductions

Managed yield tables developed for Forest Land Management Plans represent the optimum timber yields from lands suitable for intensive management. There are four situations which can result in reduced yields over time:

a. The first situation is a delay in early stand development—due to vegetation competition—that results in an extended rotation length. This may be due to a prolonged culmination of growth, or a delay in reaching a desired product size. We can achieve the same stand yields; it simply takes longer to occur in the absence of vegetation management. This extended rotation is the most significant impact in terms of potential harvest level reductions.

b. Another is tree mortality, vigor or form defects, and the suppression of growth which results in dead or submerchantable trees within the managed stand. This will translate into reduced commercial thinning opportunities and understocked areas within the stand. The disruption of normal stand development can also result in increased heterogeneity—in terms of product size and species composition.

c. Third are situations where restrictions on vegetation management options can lead to changes in the timberlands suitability classification made during the Forest planning process. Specifically, these are situations where the absence of vegetation management creates a high risk of regeneration failure. For example, this may relate to vegetation composition and structure, or physiographic conditions that produce a high risk regeneration effort in the absence of a specific management technique. The net effect on site productivity will be estimated for these conditions.

d. The final situation is a shift in species composition in response to early vegetative competition in mixed species stands. For example, relatively shade tolerant species may gain dominance within the stand in the absence of vegetation control. Factors such as growth

patterns, susceptibility to physical damage or pathogens, and reduced product values then become important. Another element of this shift in species composition can be a relative increase in hardwoods which typically have limited commercial value in the near future.

Stratification and Methodology

Six vegetative complexes will be examined in order to present a cross-section of conditions in the Region and to take advantage of the most pertinent available literature. These vegetative strata represent 55 percent of intensively managed “suitable” timberlands in the Region. The extent and geographic distribution of these strata give a cross-section of lands for the display of relative changes between alternatives in the area of timber growth and yield:

- A. DOUGLAS-FIR/RED ALDER
- B. DOUGLAS-FIR/HEMLOCK/SALMONBERRY/HERBACEOUS
- C. PONDEROSA PINE/GRASSES/HERBACEOUS
- D. DOUGLAS-FIR/PONDEROSA PINE/CEANOTHUS SPP./HERBACEOUS
- E. MIXED CONIFER/TANOAK/PACIFIC MADRONE
- F. TRUE FIR/HEMLOCK/SHRUB/GRASSES-HERBACEOUS

Analysis process steps are as follows:

Step 1. Identify growth and yield effects related to competing vegetation in each vegetative complex. This will establish a short-term relationship between a fully managed (or free-to-grow) condition and the little or no vegetation management situation normally reflected by test controls. One or several data sets will be selected as representative, and the height and diameter increment trends projected to age 15 to 20. Seedling mortality, in the absence of vegetation control, will also be estimated. Related factors such as trade-offs, competition threshold levels, and environmental factors affecting treatment effectiveness will also be examined where appropriate.

A basic reference on the subject is *Effects of Competing Vegetation on Forest Trees: A Bibliography with Abstracts*, USDA-FS, Gen. Tech. Rpt. WO-43, Sept. 1984. This is supplemented by subsequent publications, symposium and workshop proceedings, research progress reports, etc.

Step 2. The short-term growth estimates will be projected in yield simulations. This will display the per-acre yield difference between the operational site potential and the “little or no vegetation

management” situation.

Copies of yield simulation comparisons are process records on file. (USDA- Forest Service, Forest Pest Management, Portland, OR.)

Step 3. Expand the per-acre effect by an estimated total area for the display volume reductions related to the lack of vegetation management.

(Steps 4 and 5 address the relative effectiveness of herbicide use and alternative techniques under specific vegetative and physiographic conditions.)

Step 4. Identify vegetative conditions or operational factors which show efficacy differences between herbicide use and other available tools—manual, biological, thermal, and mechanical.

Step 5. If applicable, expand Item 4 by total affected area to display yield effect related to the suspension of herbicide use.

Yield model comparisons will be made with simulations appropriate to the geographic province. This will be either Stand Prognosis (Wykoff et al. 1982), the DFSIM model (Curtis et al. 1981), or Stand Projection System (Arney 1986). Rotation lengths and management schemes representative of those used in Forest Plans will be used in model comparisons. Modal site qualities for each vegetative complex will be used. On high quality sites, the percentage yield loss will be less than on low sites, although absolute volume effects may be greater (Fiske 1984).

It must be emphasized that only areas to be intensively managed—for timber yields approaching the full biological site potential—are involved in the analysis. Affected areas will be estimates because of the very simplified vegetation combinations being used. (Process records are on file.) On those lands where reduced timber yields are anticipated (to accommodate other resource values or management concerns), it is difficult to correlate the vegetation management growth effects with actual timber harvest levels.

The bridge between short-term growth effects and rotation length timber yields is weak because of growth model limitations; the need to generalize from site-specific data; and compounded error terms. However, these long-term estimates will help display relative differences in timber yields between Alternatives A, B, and C.

Douglas–fir/Alder Analysis

The humid climate within the normal alder range would indicate that light is relatively more limiting than soil moisture in this complex. Red alder is moderately intolerant of shade but will survive in an understory for several years. Full sunlight, however, is needed for normal development. Alder regeneration is commonly by natural seeding, although vigorous stump sprouting may follow site disturbance (DeBell and Turpin 1983). Uncontrolled, the rapid juvenile growth of alder can establish tree position dominance over conifers for 25 years in mixed stands (Miller and Murray 1970). Measurements in this study show dominant Douglas-fir emerging from the alder canopy at age 30.

Conifer stocking losses, due to prolonged suppression, are difficult to estimate, particularly in mixed stands where trees are initially in a codominant position. Walstad, et al. (1986) analyzed stands established in the 1950's and 1960's in which growth and species composition records could be reconstructed. Two untreated sites, converted essentially to alder, with total yields (at age 60) ranging from 46 to 62 percent of those in adjacent treated (released) stands.

Tree vigor differences after 5 years between released and unreleased trees are reported by Lauterbach (1967). In this case, suppressed Douglas-fir growing beneath alder are spindly, short and sparse-neededled, and yellowish-green in color. Mortality or submerchantable conifer in mixed stands appears to be a factor primarily in severely suppressed conditions, or a lengthy delay in conifer seedling establishment. This threshold level for seedling loss is observed by Lauterbach (1967). Cole and Newton (1986) observed conifer loss over a range of densities and levels of competition. Stein (1984) shows survival differences, at year five, in a comparison of several site preparation treatments. Lowest survival (54.3 to 64.7 percent) is reported on areas where competing vegetation was advanced at planting time. There were minor differences in a comparison of fifth-year survival on three prepared sites (70.0 to 76.2 percent) to an untreated control (64.1 percent) in the Stein study. Tree mortality does not appear to be a significant factor when reforestation is timely following site disturbance.

Growth effects, however, can be significant. Allen et al. (1978) reports a 15 percent height growth increase and 74 percent increase in stemwood volume 6 years after release of Douglas-fir seedlings on a northern Cascades site. Lauterbach (1967) shows a height growth response (after 6 years) of 6 to 178 percent over an untreated control, depending on initial seedling height. Howard and Newton (1984)

Step 1 Information Review

show overtopped seedlings to be smaller and grow more slowly than those shaded by competing vegetation. In this study, growth reductions due to tree form hardwoods—alder, maple, dogwood—were greater than those associated with encroaching lesser vegetation over the first several years of plantation development.

The beneficial effects of nitrogen enhancement in alder stands can also be a management consideration. There is much disagreement, however, regarding the trade-offs between alder competition and increased nitrogen availability. Nitrogen accretion is particularly significant during the first 6 years of alder stand development (Bormann 1977). On infertile or nitrogen-deficient sites, the trade-offs between early conifer growth suppression and soil fertility are noted in Binkley (1984). Binkley et al. (1981) examined growth and nutrition of crop trees, with and without alder, in several 22-year-old plantations. In the study, the presence of alder did not significantly affect conifer stocking levels, basal area, and average diameter and height. The conclusion was that Douglas-fir yields, when stand canopies become similar, would be greater on the alder sites because of improved tree vigor.

A similar relationship is shown in work by Miller and Murray (1970). At age 48, Douglas-fir volume in a mixed stand was 3,100 cubic feet per acre compared with 2,900 cubic feet in a pure conifer stand. Miller and Murray (1970) suggest retention of 20 to 40 red alder per acre for enhanced Douglas-fir growth on sites where nitrogen may be limiting. The Siuslaw National Forest often carries this number of alder stems in regenerated Douglas-fir stands. One possible way to take advantage of nitrogen enhancement is the introduction of alder plants after conifers are established and stocking levels are controlled, but also while site resources are still adequate for nitrogen fixation (Helgersson et al. 1984).

Nitrogen enrichment was not a factor, however, in several other studies. Cole and Newton (1986) observed no nitrogen accretion on moderately low nitrogen level sites after 5 years. Newton, et al. (1968) observed accretion only under severe soil removal conditions. The conclusion, in a report of fifth-year results by Cole and Newton (1983), is that alder growth habits appear incompatible with those of Douglas-fir in the long run, and an association of species will lead to dominance by alder and negative growth impacts.

Nitrogen enhancement, when desired, can be the result of biological fixation or through use of inorganic nitrogen fertilizers. Each of these management options have advantages and disadvantages, as noted by Carlyle (1986). The use of manipulated plant associations for nutrient cycling, rather than direct fertilization, has a strong potential. However, as noted in Carlyle (1986), further research and develop-

ment of new technologies may be required if the practice is to work consistently as a site-specific prescription.

Mixed stands of red alder and other species are more common than pure stands in most of the species range. The potential commercial value of alder is another beneficial aspect of an alder component. The low per-acre yields, high logging costs, and inconsistent markets make alder values appear minimal in the near future.

Growth comparisons and estimates used to display yield effects, due to a lack of vegetation control, are already available. The Siuslaw National Forest (Turpin et al. 1980) compared yield effects under a number of management strategies, species compositions, stocking levels, and aspects. A 28 to 29 percent yield reduction in the absence of vegetation management is estimated for "stocked conifer" sites. A rotation age of 75 years with thinnings was used under "full stocking" in this study. A typical physical rotation, with thinnings, on intensively managed sites is 80 to 90 years in current Forest Plans. This slightly prolonged growth period, in comparison to the Siuslaw study, will tend to reduce the relative effect of the early growth suppression. Therefore, the estimated yield reduction, based on the assumptions described, is 25 percent.

Stands examined in the Walstad, et al. (1986) study appear to represent the typical consequence of no or inadequate site preparation, and severe alder competition. In this case, the stands had converted to red alder at age 50, with commercial yields ranging from 46 to 62 percent in comparison to adjacent treated (released) sites.

The range of 25 to 50 percent yield falldown appears to represent a realistic range of what could be expected with no vegetation management on mixed species sites.

There are approximately 236,800 acres of this complex allocated to full yield management in the Region. A predicted reduction in long-term sustained yield levels of 25 percent is presumed for this land base.

Herbicides have a long history of use for control of alder and associated species. Manual release cutting of alder, however, has been developed and refined for improved effectiveness and duration of control. An example is the Red Alder Severing Windows Study being conducted by Washington State Department of Natural Resources (Belz 1987). In many cases, the cost-of-doing-business is now the principal differentiation. Timelines of treatment, density of alder, and the need for adequate site preparation are factors which can limit the effectiveness of any chosen technique.

Stein (1984) compared several site preparation techniques, as

Step 2 **Yield Estimates**

Step 3 **Area Expansion**

Step 4 **Herbicide** **Efficacy**

well as stock types, in a post-logging reforestation effort. Fifth-year survival was similar on both sprayed and burned units—85.6 percent versus 86.2 percent. Total height of protected trees (plastic mesh in this case) was also relatively insensitive to method. A 1984 decision analysis (Knapp et al.) examined 324 plantations on the Siuslaw National Forest for the comparison of methods for control of competing vegetation. Manual control and phenoxy herbicide use were compared in various combinations of prelogging vegetation and aspect. In all strata examined, a restriction on phenoxy herbicides had minor effects on growth (mean annual increment—MAI). The authors, however, emphasize that the study is specifically for the Siuslaw National Forest, and that the results may not be applicable elsewhere.

The Olympic National Forest has a large (111,000 acres) area of alder influence sites being managed intensively. The Olympic has relied primarily on manual treatments for alder control in the silvicultural program. A similar pattern has been shown in the conifer-alder component on Forests in the northern Cascades province.

Step 5 Herbicide Yield Effects

Based on information shown, plus assumptions used in this assessment (i.e., timely action following site disturbance and the disregard of treatment costs), the yield effects, due to method effectiveness differences, appear to be minor.

Turpin (1987) identified sources of new information and methodology which has helped the Siuslaw National Forest deal with an absence of herbicide use since a 1983 U.S. District Court of Oregon injunction against their use. These factors are:

1. The development of higher quality seedlings and animal control techniques with an effect of giving plantations a fast start and reducing the necessity for replanting.
2. Target stocking levels have been reduced as a result of better yield and economic models. The desired stocking at year 10 is now 250 to 300 trees per acre, rather than the previous 430 trees per acre.
3. Forest-specific research to develop “windows of success” for hand release of red alder. Following the developed guidelines has given the Forest a success rate of 96 percent for hand release.
4. Treatment needs in a manual release effort can be confined to only that fraction of an area actually in need of release. Aerial herbicide application is by nature a more broad scale operation.

Douglas–fir/Hemlock/Salmonberry/ Herbaceous Analysis

Step 1 Information Review

Salmonberry and its associates (vine maple, thimbleberry, elderberry, alder, salal, etc.) have the ability to aggressively reoccupy a site following disturbance. Wagner (1984) reports that brush encroachment exceeded pretreatment levels within 2 years after manual cutting on salmonberry-dominated sites of the Coast Range. In second-year measurements, Roberts (1980) found treated areas recovered to 85 percent of pretreatment levels following manual cutting.

Moderate to heavy volumes of this brush complex can cause competition for moisture and light for overtopped seedlings. A species composition index is being developed for the Siuslaw National Forest (Wagner and Radosevich 1986). When found in abundance, salmonberry and several other shrubs with rapid early growth (such as thimbleberry, vine maple, and hazel) were considered important during the first several years of plantation development. After this period, they were felt to decrease in importance, as the Douglas-fir surpasses them in height.

Early planting of a prepared site appears to be particularly important when dealing with salmonberry and associated species. Newton and White (1983) found significant differences in seventh-year survival for all stock types planted initially in 0- or 4-year-old brush. Survival of released seedlings was 75 percent, compared to 38 percent for the unreleased trees initially planted in 4-year-old salmonberry. In this case, mortality also added substantially to the number of years expected before stand crown closure. Stein (1984) shows slight improvements in fifth-year survival in a comparison of several release treatments. However, in two other studies by Stein (1986), and previous work by Ruth (1956), there are no significant correlations between vegetation control and early seedling mortality. Third-year results showing Douglas-fir growth and survival following release treatments (on salmonberry-dominated sites) are reported by Harrington and Wagner (1986). The study sites were prepared (burned or sprayed) and released 2 to 3 years after planting (chemical or manual treatments). Douglas-fir survival was not significantly different among treatments after 3 years, including both the complete removal site and an untreated control.

Seedling survival on salmonberry influence sites appears to be directly related to adequacy of site preparation and timely reforestation. Tree mortality is consistently minor when conifers are established in a competitive crown position with the shrubs. The Newton and White (1983) data appears to be a reasonable proxy for what would

result under severe salmonberry competition. However, for this analysis no significant mortality will be displayed for the “no vegetation management” situation.

The short-term studies referenced above indicate reduced seedling growth in the absence of vegetation management. While both seedling height and diameter increment effects are seen, diameter growth appears to provide the most consistent correlation between salmonberry control and a release effect. Factors such as physical or chemical damage to individual tree leaders during release, animal browsing, “shock” effects, or differential tree growth in response to competition (for example, etiolation—increased height growth at the expense of diameter growth) can temporarily set back the height response. Stein (1986), in a research progress report, finds significant differences in diameter growth after 4 years between treated sites and a control. However, no differences in height increment are observed. Released sites in this study show a 50+ percent increased diameter increment after 4 years; an average of 53.7 millimeters (mm.) for treated sites, compared with 35.5 mm. for a “no release” site.

A study reported by Harrington and Wagner (1986) indicates growth trends under several release treatments. Diameter increment, in this case, was significantly greater after 3 years only under a “complete removal” treatment. Comparison of more operationally realistic prescriptions (one manual and three different herbicide-use sites) shows no significant diameter or height increment differences when compared to an untreated control.

Seedlings planted in well established salmonberry, however, can suffer severe growth reduction and vigor loss. Newton and White (1983) found each year’s delay in planting of salmonberry threat sites compounded losses from competition when compared to a site vacant of brush. Competition added .4, 1.5, and 4.1 years to the time required to reach a height at which trees may be considered “free-to-grow” from plantings in 0-, 2-, and 4-year-old salmonberry, respectively.

Several studies have been designed to show the relative increase, or encroachment, of herbaceous vegetation on released salmonberry sites. The removal of salmonberry and associated species will encourage an increase in annual plants and grasses. In this situation, on sites not subject to a severe summer drought and moisture stress, weed and grass control is generally not needed to establish Douglas-fir plantations. On interior Coast Ranges and Cascade Range sites, however, lack of timely herbaceous vegetation control has been shown to translate into adverse survival and growth effects by both Gratkowski et al. (1979) and Preest (1977).

The Stein (1986) Research Progress Report (FS-PNW-1201-8029) appears to represent a typical severity of salmonberry competition.

The average height and diameter increment trends in this study will be projected for comparison of effects due to the absence of vegetation management.

Reforestation of salmonberry influence sites can be complicated by animal damage problems that are directly related to the intensity of vegetation management. The failure to control both vegetative competition and habitat for mountain beavers, rabbits, or hares, can result in poorly stocked holes in young stands.

Perry et al. (1985) identifies the need for more knowledge of both salmonberry growth patterns and competitive effects on conifers. Aggressive site occupancy and exclusion of conifers by species such as salmonberry, thimbleberry, bracken fern, and vine maple has been observed. However, with current state-of-knowledge, it is difficult to establish threshold values for significant competitive effects to conifers.

A 90-year rotation with intermediate stand entries is representative for intensively managed salmonberry influence sites.

Using the Stand Project System model for yield projection, the volume loss associated with an absence of vegetation management is approximately 21 percent.

There are approximately 195,100 acres of this vegetation type allocated to "full yield" management in the Region.

Few studies have been designed to show the relative effectiveness of herbicides and nonchemical methods in the control of salmonberry. Manual cuttings, glyphosate, fosamine, and a manual-plus-fosamine treatment are being analyzed on Coast Range sites by Stein (1986). Intermediate results after 4 years indicate no significant differences in chemical or manual release in terms of survival and growth. A Coast Range Site Preparation Study Progress Report by Stein (1986) indicates no significant difference between fifth-year survival in manual, chemical, and broadcast burn treatments under a moderate level of brush competition to protected (tubed) seedlings. In both height and diameter growth, trees in broadcast burn treatments showed greater gains than for the other treatments. In a companion study, Manual and Chemical Options for Releasing Douglas-fir (Stein 1986), fourth-year results indicate that release treatments had little effect on tree survival or height growth, but some significant effects on diameter growth. In this case, three intensities of manual cutting showed the highest percentage of diameter increment gain (484 percent) compared to aerial glyphosate (461 percent) and fosamine (465 percent) applications.

Step 2 Yield Comparison

Step 3 Area Expansion

Step 4 Herbicide Efficacy

Harrington and Wagner (1986) report third-year growth and survival following three chemical and a manual treatment on Oregon and Washington Coast Range sites. This study was conducted in young brushfields, with only moderate amounts of overtopping brush. None of the operational release treatments (glyphosate, triclopyr, or manual cutting) have significantly increased Douglas-fir height or stem diameter growth above the untreated control.

With limited information, there is insufficient evidence to indicate growth and yield differences between vegetation management techniques in the control of salmonberry competition. Use of herbicides, particularly glyphosate, has proven an effective tool for dealing with excessive salmonberry and herbaceous vegetation. However, under the assumptions used in this assessment (cost is not limiting, and reforestation following site disturbance is done in a timely manner), there appears to be no significant differences in treatment effectiveness.

Step 5 Area Expansion

No significant yield reduction due to the suspension of herbicide use can be shown for the salmonberry type.

Ponderosa Pine/ Grasses/Herbaceous Analysis

Step 1 Information Review

This is a broad model for a vegetation complex that includes some diverse plant associations in eastern Oregon and Washington. Ponderosa pine generally predominates in association with species such as lodgepole pine, western larch, Douglas-fir, white pine, or true firs. Shrub species such as manzanita and bitterbrush can often be important components. There is a sizable body of information dealing with pine establishment on grassy sites in the Pacific Northwest, and more generally, in the entire Western United States. For this reason, the pine-grass complex will be used as a broad proxy for these diverse vegetative conditions. Another general characteristic of this type is the relatively low site quality in comparison with the other complexes being analyzed.

Several studies have documented both mortality and reduced tree vigor on drought-prone sites with the absence of grass and forb control in new plantations. In a 10-year trial by Crouch (1979) in south-central Oregon, two fall herbicide applications doubled survival (55 percent versus 25 percent) and significantly increased heights (222 centimeters (cm.) versus 150 cm.) of released pines. Several shorter term results by Baron (1962), Christensen et al. (1974), Dimock et al.

(1983), and Bickford (1965) have shown stocking reductions. A Tahoe National Forest administrative study of grassy, unreleased pine plantations is reported by Fiske (1984). Average trees per acre stocking in these 0- to 20-year-old stands was 68, with only 48 percent of plots meeting minimum acceptable stocking standards. Seedling survival tends to vary greatly in response to spring and early summer precipitation patterns for any given season. Tree mortality, due to weed and grass competition, appears to be particularly responsive to the severity of competition.

At high elevations, the early control of herbaceous vegetation in reforested units will remove the food supply of pocket gophers, which can result in reduced tree mortality. When used at the proper scale and time, grass/forb control can effectively limit plantation damage due to rodent populations (Crouch 1979).

With no vegetation control in this complex, the 50 percent seedling loss within the first 5 years in the Crouch (1979) study appears to be a representative figure. Under intense grass and weed competition, however, 70 to 100 percent mortality figures have been reported in annual plantation monitoring surveys conducted by the East-side Forests.

Annual and perennial grasses have the potential to quickly occupy a large volume of soil and to initiate root growth earlier in the spring than competing conifer seedlings (McDonald 1982). On sites where soil moisture is limiting, seedling vigor may suffer for extended periods until an adequate rooting depth is reached by the conifers. Diameter growth reduction in the absence of early grass and forb control has been reported by Gordon (1962). This study in sapling size pine shows a potential 39 percent increase in radial growth after 5 years. Crouch (1979) shows a possible 38 percent greater height response at 10 years with timely grass/forb control. Fox (1985), in a progress report, displays ninth-year results in a 25-year-old pine stand with a dense understory of snowbrush, manzanita, and chinkapin on the Winema National Forest. Cubic volume underbrush control is 38 percent greater (1,022 cubic feet versus 788 cubic feet) in comparison with the untreated control.

The threshold level for competitive growth and mortality effects due to grass density is displayed by Petersen (1985). At low levels of grass density (Leaf Area Index), competitive stress is apparently sufficient to reduce stemwood growth of some trees, but not severe enough to cause mortality. This is attributed to the ability of ponderosa pine to change patterns of photosynthate allocation from stemwood production to root production in order to tap water stored at deeper soil layers. Under severe competition, however, Peterson implies that substantial reductions of herbaceous cover are needed

before resources are made available to alleviate the density-dependent stress in seedlings.

The study by Gordon (1962) displays growth suppression likely to be shown in grassy pine plantations. This study shows a potential 39 percent increase in radial growth after 5 years, and Crouch (1979) indicates a 38 percent greater height response at 10 years with timely grass and forb control. These growth trends will be projected to display short-term effects due to an absence of vegetation management.

Step 2 Yield Effects

Yield comparisons were made with Prognosis—the SORNEC variant (Johnson et al. 1986). A 100-year rotation with two intermediate entries is typical of intensively managed pine stands. Using the assumptions described, no vegetation management will result in a 52 percent per acre yield reduction.

Step 3 Area Expansion

Approximately 1,436,200 acres of the pine-grass community is allocated to “full yield” timber management. The predicted 52 percent change in long-term sustained yield on this land base is assumed in the absence of vegetation management.

Step 4 Herbicide Efficacy

In plantations with severe grass and forb competition, herbicide unavailability will translate into a yield effect. Effective site preparation can be obtained through a variety of methods in this vegetative type, or where steep slopes are seldom a limiting factor. Plantation site preparation and release for survival or growth has generally been accomplished through herbicide use, manual weeding and grubbing, and the forced grazing of cattle.

Reinvasion of new plantations by grass and weeds is highly variable on sites east of the Cascade Crest. The potential for rapid site occupancy, particularly by annual grasses and sedges at higher elevations, has been documented by several authors, including Gordon (1962) and McDonald (1982). Grass species differences have been shown to be an important factor in tree seedling moisture stress (Baron 1962), both because of grass growth qualities and the species selectivity in grazing by cattle.

The need for release from grass-forb competition is related to the amount of competing vegetation. A threshold level for effects due to grass competition in ponderosa pine plantations has been indicated in work by Peterson (1985) and others. An estimate, based on experience of several East-side Forests, is that approximately 20 to 30 percent of plantations in the pine/grass community will require a release treatment, as well as site preparation, for acceptable early stocking and growth.

On sites where grasses are well established, spot scalping or grubbing will not control moisture competition in subsurface soils due to the extensive grass root systems. This means that a complete area treatment would be necessary for the desired release effect in these situations. Even if treatment cost is disregarded, large-scale, full-area grass removal in existing plantations is unlikely to be operationally feasible. Alternative methods, such as prolonged or forced animal grazing, can be effective. However, limitations imposed by herd or band availability—and the large administrative impact of these projects—tend to militate against a widespread reliance on grazing for plantation release programs.

It is estimated that an operational release program using manual and biological methods could accommodate approximately two-thirds of the grass control needed to release intensively managed pine plantations. This means that approximately 478,700 acres would be untreatable in the absence of herbicide use. For modeling purposes, a management scheme similar to that used for “no vegetation management” has been used. In other words, a failure to use herbicides on these particular acres will have the same consequence as “no vegetation management.”

A simple proportion is used to estimate yield reductions in the absence of herbicide availability in grassy-pine plantations. Based on operational constraints described in Step 4, roughly 8 percent of acres in need of plantation maintenance will be untreatable in the absence of chemical release ($33\frac{1}{3}$ percent untreated \times 25 percent in need of release). This means that a potential reduction in long-term sustained yield of 4 percent will be associated with a suspension of herbicide use. (8 percent of area untreatable \times 52 percent yield reduction due to no vegetation management.)

On a Regionwide basis, under the assumptions used (substitutability of techniques; cost factors nonlimiting; and timely reforestation following site disturbance), the lack of chemical use in this vegetation complex would involve only modest yield reductions. This effect can be significant locally, however, on several individual Forests within the pine subregion.

Step 5 **Yield Effects**

Douglas-fir/Ponderosa Pine/ Ceanothus Spp./Herbaceous Analysis

Step 1 Information Review

Snowbrush (*Ceanothus velutinus*) is a widespread and silviculturally important component of many conifer stands. Deerbrush (*C. integerimus*) is locally important in southern Oregon and has been evaluated in several studies. Both species can be aggressive sprouters following site disturbance, and can maintain large seedloads in litter and surface soils.

Site reoccupancy is normally the result of old seed stored in the soil. For this reason, effects on conifer seedling survival can vary greatly, depending on treatment or disturbance of stands influenced by these shrubs. For example, in deerbrush of wildfire origin (severe site disturbance), McDonald and Fiddler (1986) found differences in fourth-year survival—89 percent versus 42 percent—between released and control conifer plots. On sites more representative of normal timber management regimes, early stocking losses due to ceanothus competition have not been severe. Gratkowski and Lauterback (1974), and Stein (1986) either found tree mortality to be insignificant or related mainly to factors such as animal damage.

Walstad et al. (1986) compare treated (released) and untreated (*C. velutinus*) influence sites which were logged in the 1960's. Douglas-fir stocking and diameter growth were reduced on the untreated site. Growth simulations (DFSIM) with final harvest at age 60 indicate at 13 percent volume reduction—9.9 cubic feet/acre (c.f./ac.) versus 11.4 c.f./ac.) in the unreleased stand. Product value, expressed as net stumpage values, show a large proportionate difference between stands (\$13,700/Ac. versus \$9,200/Ac.) in this study.

Growth and form effects, due to moderate or severe competition, are well documented.

Early work by Dahms (1950) in snowbrush and manzanita shows significantly reduced height growth in unreleased seedlings. Height response, however, has tended to be inconsistent because of shock effects, physical leader damage, or animal browsing after release. Gratkowski (1979) found most of the height increment response to occur in the second 5-year period following treatment. Trees initially 8 feet tall at time of release in this study showed 1.2 to 1.4 times the growth of the trees growing in mature snowbrush. Gratkowski and Lauterbach (1974) estimate that release from snowbush on their study sites will save 8 years in reaching a 20-foot average tree height.

Diameter increment has shown to be a more reliable measure of competition and response in work by Gratkowski (1979), Peterson and Newton (1982 and 1985), Chan (1985), and McDonald and Fiddler

(1986). Fourth-year caliper of seedlings growing in the "entire area sprayed" were approximately 2.5 times that of the control in McDonald and Fiddler (1986). Gratkowski (1979) indicates a 40 percent increase of radial growth at 10 years in the full exposure treatment (cut and stump-spray).

Peterson and Newton (1985) display the importance of dealing with both the shrub and herbaceous components. Fifth-year data shows significant increases in stem height, diameter, and stem volume when snowbrush and forbs were sprayed—only a diameter increment response was shown when shrubs alone were treated.

Definition of a typical severity of ceanothus competition is difficult due to the highly variable site limitations, stand histories, and brush densities displayed in the research. For example, extrapolation of results from northern California studies to the Central Cascades, where soil moisture is normally less limiting, can lead to an over statement of growth and yield effects.

It has been shown that competitive effects of snowbrush and associated species can linger long after trees gain dominance in terms of crown position. Working in a thinned 12-year-old stand, Oliver (1984) found significant differences in fifth-year response when full brush removal was employed. This was on a relatively low productivity site dominated by whiteleaf manzanita, a nonsprouting variety. Response, after 23 years, in suppressed 40- to 70-year-old saplings was found to vary significantly with snowbrush and manzanita control, following overstory removal and thinning in Barrett (1982). The importance of early brush control is reinforced by several authors. Peterson and Newton (1982) conclude that trees that have grown in competition for as long as 10 years will show little early response to release from snowbrush. The need for early snowbrush control in conifer plantations is also indicated in studies by Youngberg and Wollum (1976), and Gratkowski (1979). A consistent message from the literature seems to be that "the longer the suppression, the longer it takes for release."

Tree vigor and form effects related to competition from snowbrush and associated species are addressed in the literature. Gratkowski (1979) finds trees shaded by a dense shrub canopy to usually be slender-stemmed and fragile, with narrow crowns and sparse foliage. Seedlings growing on a deerbrush influence site were found by McDonald and Fiddler (1986) to sacrifice stem caliper and live crown development for height extension. Horowitz et al. (1978 and 1982) addressed an apparent anomaly of increased height growth when seedlings were growing near ceanothus plants. Sites in this case were selected at random from units scheduled for release. Many trees in these studies were found to be healthy when growing in the presence

of brush, and the need for release of treated units was therefore questioned. An observation is also made from the site-specific cases that enhanced soil nitrogen availability in ceanothus brush fields, and the potential for damage to nontarget vegetation make release through herbicide use of questionable value. In addition, many trees in these units were found to simply not be suppressed. This study by Horowitz has been questioned on the basis of covariation among the comparisons—that a mistake was made in confounding conifer location with site history. The general observations are useful, however, because of the large number of sites examined.

The question of trade-offs between control and the beneficial aspects of the ceanothus component involves much uncertainty. Net effects over a full rotation are not certain. The relative value of biological nitrogen accretion on nutrient-deficient sites, compared with application of inorganic fertilizers, has likewise been poorly defined.

Walstand (1986), however, finds the importance of nitrogen fixation on ceanothus species sites to be not well understood. Conard et al. (1985) concluded that there is little proof that nutritional benefits would compensate for a growth loss due to brush competition. As with alder nitrogen accretion, the benefits from ceanothus species will probably be important on nutrient-deficient sites and of limited value on others. A moderate stocking level of snowbrush stems is often considered advantageous in operational stand prescriptions in the Pacific Northwest Region. In the absence of documented long-term information, the bulk of available literature appears to indicate that the typical net effect of a large shrub component in young plantations will be detrimental to conifer yields.

Conifer growth effects due to moderate and severe snowbrush competition can be persistent in comparison to some other shrub/forb complexes. This is particularly true of the variety *C. velutinus* v. *velutinus*, which can grow to a height of 9 to 10 feet. It is felt that the Gratkowski (1979) data for trees initially tall (8 feet in height) at time of release are representative of moderate levels of ceanothus competition.

Height and diameter increment trends over 10 years in this study will be projected for short-term effects due to a lack of vegetation management. Diameter increment trends from the McDonald and Fiddler (1986) study will be used to display a severe level of ceanothus competition. The extent and location of this brush complex within the region ensures that fire rehabilitation and regeneration of older brushfields will be necessary. This is expected to be the case in approximately 15 to 20 percent of the reforestation efforts. Potential seedling mortality will, therefore, be assumed on a proportionate basis. The typical short-term conifer mortality is therefore 10 percent

(50 percent mortality in stands of fire origin (from McDonald and Fiddler (1986)) x 20 percent of the total reforestation effort).

A representative physical rotation, with commercial thinning yields, is 90 years in this complex.

Yield simulations indicate a 39 percent yield reduction due to an absence of vegetation management.

Approximately 912,600 acres of ceanothus influence sites are allocated to "full yield" management.

Several manual and mechanical techniques have proven to be operationally effective in dealing with these seral shrub species. Unless root crowns are removed during mechanical site preparation, however, prolific resprouting may occur. Vigorous regrowth also can result following manual cutting, although the pulling of ceanothus germinants and young plants is effective in very young stands.

The principal advantage of the use of systemic herbicides in ceanothus species control is the inhibition of the potential rapid site reoccupancy, and therefore creation of a more manageable plantation maintenance situation. This difference in the duration of effectiveness is probably significant only on fire rehabilitation sites, or where early shrub control has not been employed.

These situations will result in an increased severity of competition. Indications of this can be seen in McDonald and Fiddler (1985), where significant differences in seedling survival and stem caliper after 4 years is seen among three manual and a chemical treatment in deerbrush of fire origin. Gratkowski (1979) also finds that tenth year radial growth differences can be seen in herbicide (aerial) and manual (cut and resprout) treatments among seedlings that are initially short in relationship to competing snowbrush plants. In both studies, the radial increment in manual treatment plots shows roughly half of the response seen in the herbicide treatments. Conifer height growth, however, appears to be relatively unaffected.

Relative changes such as this (a constant height growth, decreasing radial growth, and minor tree stocking reductions) are difficult to display in a stand projection model. For this reason, yield effects related to an inability to use herbicides, will be estimated using the "no vegetation control" adjustment in Step 2 as a reference. It is emphasized that this difference in treatment effectiveness will apply only on those sites where well established older brushfields exist or fire rehabilitation is being attempted.

Based on the majority of these studies, early mortality differences are probably minor between herbicide and manual treatments in

Step 2 Yield Effects

Step 3 Area Expansion

Step 4 Herbicide Efficacy

the ceanothus species type. Growth effects related to the unavailability of herbicides, however, will be severe on the 15 to 20 percent of all ceanothus sites where timely release efforts have not taken place (see Step 1).

A diameter growth response to release from ceanothus species is commonly seen in the literature described in Step 1. Height response, however, has been inconsistent.

In many tree volume equations periodic diameter increment contributes approximately twice as much as periodic tree height growth to cubic stem volume in stands approaching typical rotation ages. Examples can be seen in managed yield tables shown in Curtis et al. (1982). Using simple proportions, therefore, the volume reduction associated with herbicide loss is estimated to be 10 percent of that due to a total lack of vegetation control (17-1/2 percent of acreage, times a two-thirds volume influence related to diameter change, equals 11.7 percent, or roughly 12 percent of the adjustment shown for "no vegetation management" in Step 2).

Step 5 Area Expansion

The predicted change in long-term sustained yield related to a loss of herbicide use on ceanothus influence sites is, therefore, a loss of 5 percent. (39 percent adjustment for "no vegetation management" times 12 percent) There are approximately 912,600 acres of ceanothus influence sites allocated to full-yield management.

Douglas-fir/Tanoak/Madrone Analysis

Tanoak, Pacific madrone, and associated tree and shrub species can present severe site competition to conifers in southwestern Oregon and northwestern California forests. A number of environmental variables are common within the Interior Coast Ranges and Siskiyou Mountains which, in combination, create the potential for severe moisture stress to conifer seedlings. These include the severe summer drought, soils which can be of limited moisture holding capacity due to depth or texture, and generally steep terrain. White (1985) identifies factors such as thin skeletal soils, hot dry summers, and aggressive herb and shrub competition as contributing to difficulty in establishment of conifer plantations in southwest Oregon. Minore et al. (1984) correlated environmental variables with regeneration success; finding soil depth, percent coarse fragments, history of slash burning, and a southerly aspects most often associated with poor regeneration.

Both tanoak and madrone develop quickly as seedlings, and particularly, as vegetative sprouts. After three growing seasons, Strothmann and Roy (1981) found sprouts to average 7 to 10 feet in

height and occur in clumps of 12 to 13 stems. The species can achieve tree form with mature individuals generally 50 to 90 feet tall, and individual tanoaks can grow to 120 to 150 feet in height on high quality sites.

Strothmann and Roy (1981) also address the potential severity of tanoak and snowbrush competition. Twelve years after logging, some individual snowbrush plants had begun to die. Tanoak, however, was providing increasingly severe competition. The potential severity of tanoak competition is also illustrated by Tappeiner (1985). In this case, tanoak potential site occupancy (measured in Leaf Area Index) is high in comparison to Ceanothus and white-leaf manzanita.

While aggressive site reoccupancy is normally the result of vegetative sprouting, both species produce copious amounts of seed at frequent intervals (McDonald 1987). Few of these seeds result in trees, but enough do to assure that hardwoods are a continuous component of the understory vegetation. Models have recently been developed for prediction of site occupancy by sprouting hardwoods (Harrington et al.). This ability of broadleaved sclerophyll species to produce new leaf surface area by sprouting following mechanical or fire damage contributed to the relatively high competitive ability of these species.

There is little information available regarding seedling mortality, due to long-term tanoak competition. Roy (1975) addressed tree vigor and crown position relative to brush canopy of the twinned trees after 17 seasons. 55.6 percent of the brush-grown trees are classed as vigorous, compared to 100 percent of the open-grown seedlings.

Relative tree position shows a similar relationship, with 51.9 percent of brush-grown conifers in a dominant or codominant position, compared to 92.6 percent of the open-grown trees. There was also an especially high mortality in trees growing through the brush due to snow breakage. This factor may be related to the reduced vigor and form defect (etiolation) common in Douglas-fir grown under severe competition. The ability of individual tanoak and madrone to maintain height growth for long periods probably means that few of the over-topped trees will survive and maintain adequate growth for the production of a commercial product.

The Brush Mountain Twinning (Roy, 1975 and 1981) represents a typical degree of competition and site history in tanoak influence sites. For this reason, a 40 to 50 percent seedling mortality is assumed in the absence of vegetation management. The study represents a direct comparison of trees growing in a brush-free area and a brush-grown situation. Reforestation efforts following site disturbance (logging) were made promptly in both situations.

The Roy (1975 and 1981) data addresses height growth effects. After 28 years, the average "brush-grown" trees had lost 12.4 feet in

height, representing a loss of 8.3 years growth. Differences in height growth were slow at first, but the growth curves were continuing to diverge after 28 years. The Roy data, in fact, indicates that release effects will not be apparent until 5+ years following release. Vigor differences—expressed as needle complement, color, and needle length—were apparent in brush-grown trees. After projecting the growth curves from this study, Fiske (1983) estimates a 12- to 13-year delay (40 years compared to 27 years) for the suppressed trees to reach 50 feet in height. This seems consistent with interim growth estimates made by Tesch (1985), after observation of seedlings growing in tanoak and manzanita sprouts. In this case, it would take approximately 25 years for seedling in a hand-slashed area to reach breast height, while Douglas-fir on a complete control site, should reach the same height in less than 8 years.

Diameter response has been reported in work by Owston et al. (1986). Fifth-year results from a Coastal portion of the Siskiyou National Forest show a significant diameter growth increase (15.5 mm. versus 10.9 mm.) of trees in chemically treated plantations. Radosevich et al. (1976) evaluated Douglas-fir radial growth for 10 years following a cut-surface herbicide control of tanoak and madrone. Trees receiving an overstory control showed a 260 to 451 percent increased basal area growth in comparison to controls. Third-year height and diameter response in bare-root seedlings growing under injected hardwoods (Hobbs et al. 1986) was 70 to 80 percent greater than those grown under the untreated hardwoods.

The Owston et al. (1986) data appears to represent a typical severity of competition and will be projected to display short-term radial growth effects.

Projections of height growth response by Fiske (1983) based on the Roy data will be used to display short-term consequences of an absence of vegetation management.

Work by Tappeiner and Harrington (1985) reinforces the need for control of both herbaceous and shrub components in release efforts. Third-year results demonstrate an increase of stem diameter growth only with complete removal of shrub and herbaceous vegetation. The importance of herbaceous vegetation control during the early seedling establishment period is also implied in preliminary results by Hobbs et al. (1986).

Tree form tanoak have commercial value as a roundwood product, and bark of some older trees is utilized for its tannin content. Hardwood markets in the near future are limited, however, and tend to be directly related to the availability of sawmill residues for use in pulping operations.

A 90- to 100-year rotation, with commercial thinning, is representative of intensively managed stands is this complex. Comparison of growth projections using DFSIM indicate that the yield reduction associated with no vegetation management is 60 percent over a typical management cycle.

Step 2 Yield Comparisons

There are an estimated 169,300 acres of this vegetative complex allocated to full yield management on the Siskiyou National Forest and minor portions of the Rogue River and Umpqua National Forests.

Step 3 Area Expansion

A large portion of the total historic herbicide use has occurred in the tanoak/madrone belt. Both species can sprout vigorously following fire or soil disturbance. Tappeiner and McDonald (1984) found tanoak seedlings to maintain a single stem for about 5 to 12 years, then form a burl with dormant buds below ground and produce new stems. The study also indicates that tanoak growing in an understory reestablishes slowly after burning.

Step 4 Herbicide Efficacy

The implication is that tanoak control in 30- to 75-year-old conifer stands may effectively prevent the need for costly subsequent site preparation. Mechanical removal of large root crowns is difficult without major site disturbance. The tough, leathery foliage discourages any significant browsing of tanoak, although the berries (madrone) and acorns (tanoak) are utilized by wildlife. The current season's growth of madrone is occasionally browsed by deer.

Manual release using power saws has shown mixed results. After working with canyon live oak, a common associate on relatively dry sites, Hobbs and Wearstler (1985) concluded that manual slashing of sclerophyll brush is not operationally realistic due to the vigorous resprouting and potential for physical damage to crop trees during the felling of hardwoods. Forest Service silviculturists in southwestern Oregon and northwestern California are having some success with manual tanoak control, although more than one treatment is often needed to meet release objectives.

Herbicide release treatments must be timed precisely to accommodate differences in phenological development of target and non-target vegetation (Gratkowski 1978, and Conard 1983). When properly used, however, herbicides have proven effective in both the degree and duration of tanoak and madrone control. The principal advantage of systemic herbicide use in tanoak control lies in the inhibition of vegetative sprouting in comparison with mechanical or manual treatment. Siskiyou National Forest records indicate that 1.5 chemical treatments have typically been used to meet prescription objectives (plantation certification). An average of two to three manual treatments, however, have been necessary to achieve comparable results on

tanoak influence sites.

For reasons stated above, the amount and vigor of preexisting tanoak is a major factor in the relative effectiveness of herbicide and alternative methods. It is doubtful that conversion of pure or nearly pure tanoak and madrone stands to conifer plantations will be successful (i.e., meet the tree survival and growth rates assumed in managed yield projections) without chemical brush control to limit resprouting.

The same limitation would apply in the regeneration of poorly stocked conifer stands, with a well established tanoak component. This means that an estimated 50,000 acres of tanoak-dominated sites cannot be successfully regenerated in the absence of herbicide use. (Based on a survey of silviculturists on the Siskiyou, Rogue River, and Umpqua National Forests).

The yield effects related to a lack of vegetation control, shown in Step 2, are considered a reasonable proxy for efforts to regenerate these 50,000 acres of severe hardwood competition by nonchemical methods.

Based on assumptions used in this analysis (i.e., cost is not limiting, and reforestation is timely) chemical or manual treatments should be effective where conifer was dominant in the original stand (in terms of crown closure).

At this time, there is insufficient data to quantify differences between methods. The current prescription for manual cutting of tanoak on the Siskiyou National Forest is limited to a single treatment after conifers reach a mean height of 36 to 48 inches. This is a reflection of concerns for costs factors, buildup of fire fuels, and worker safety when working in heavy slash.

Studies being conducted by the Forest Service, Pacific Southwest Forest and Range Experiment Station, and the Oregon State University FIR (Forestry Intensified Research) Program are examining the effectiveness of various nonchemical controls of tanoak competition in conifer plantations. Knowledge gained in this research, plus the continuing efforts of Forest Service and other silviculturists, should help refine the stand culture and harvest techniques needed to meet management objectives in the tanoak complex.

Step 5
Area Expansion The loss of herbicide use would result in approximately a 19 percent reduction in predicted long-term sustained yield.

(Tanoak conversion area divided by total tanoak area, times yield reduction due to "no vegetation management.")

True Fir/Hemlock/Shrub/ Herbaceous Analysis

Regeneration success has been variable in these higher elevation stands due to extremes in solar radiation, air temperature, and moisture stress, which can be intensified by brush, grass, and herbaceous competition. Tree damage by rodents feeding in sedge and forb communities can also complicate the reforestation efforts. True firs (Pacific silver, subalpine, noble, grand, red, or white fir) are normally important components in a variable mixture of montane and subalpine tree species. Unfortunately, there is little information regarding competing vegetation beyond the seedling stage in this vegetation type. Once established, hemlock and most true fir seedlings and saplings have the ability to maintain full crowns and the potential for a favorable growth response despite long periods of suppression. This advantage has been documented by Seidel (1983) and others. A variety of silvicultural systems and vegetation control techniques are being used in management of true fir.

After examining clearcuts in the East-side Cascades, Seidel (1979) identified an increase in grasses and forbs (along with the severity of burning) to be consistently associated with decreased tree stocking. Dimock (1981) has found significant increases in third-year survival of true fir when sedges and beargrass were controlled in herbicides—26 percent and 31 percent respectively, compared with 9 percent and 14 percent on control plots. Working with established white fir understory saplings in the Central Sierras, Conard and Radosevich (1982) indicate that a combination of dead shade and brush control can be a benefit to survival of true fir naturals because of the reduced evaporative stress. When no shade was provided, at least an 80 percent reduction of shrub cover was needed before growth effects were recorded.

In another Central Sierras study involving mixed conifers and montane shrubs, Lanini and Radosevich (1986) found white fir survival at year 5 to range between 41 percent and 52 percent on sites receiving mechanical or thermal site preparation. Survival on plots where herbicide applications had been combined with the site preparation treatments were consistently lower, due to the accidental exposure of sensitive fir seedlings during application. Tolerance to herbicide exposure varies greatly by tree species, type of herbicide used, and physiologic factors related to seasonal growth patterns (King and Radosevich 1985).

The number of potentially limiting environmental variables

Step 1 **Information** **Review**

and variety of silvicultural schemes being used in the true fir-hemlock type make the quantification of tree survival and growth effects relatively uncertain. Often, the most competitive vegetation on these sites occurs in the early seral stage. This means that severe site disturbances, such as broadcast burning or machine piling of brush, can result in subsequent vegetation management and productivity problems.

True fir survival, in particular, appears to be related to control of grass-forb competition during the critical period of seedling establishment. A reduced tree per acre stocking level can be especially damaging to long-term volume yield on these sites which are capable of maintaining high stocking levels and favorable growth rates for long time periods. Using data from Dimock (1981), it is estimated that seedling survival on sites receiving no vegetation management will be 35 to 45 percent of that on sites with early weed and brush control.

White fir height and diameter growth response in the King and Radosevich (1985) study was related to reduced shrub canopy volume. In this case, the "low" shrub canopy volume sites show a 22 percent larger height (38.0 cm. versus 31.2 cm.) and 57 percent greater diameter (13.5 mm. versus 8.6 mm.) response in comparison with fir on the "high" plots. The herbicide applications generally increased the available soil water over the nonchemical-treated plots, regardless of site preparation methods (mechanical or thermal) used in combination.

Data on seedling growth effects, however, is simply too limited for use in the development of short-term trends. There is a particular lack of data showing fir growth effects on Central Cascades sites. In the Northern Cascades, the high elevation true fir-hemlock type generally lacks a significant shrub component and has therefore not been included in this analysis.

Step 2 Yield Effects

A 120-year-rotation, with intermediate thinnings, is representative for this type. The tree stocking effects (a 55 to 65 percent loss from Dimock 1981) were used to display yield effects in growth simulations. An absence of vegetation management would result in an approximate yield reduction of 56 percent in comparison with full yield management.

Step 3 Area Expansion

Approximately 1,474,700 acres of this vegetation complex are allocated to "full-yield" management in the Pacific Northwest Region.

Step 4 Herbicide Efficacy

Herbicide use has shown effectiveness in operational programs for the control of shrubs, forbs, and grasses in young true fir stands. This is especially true when intertwined root systems of seedlings and sod-forming grasses preclude the use of manual grubbing and scalping.

Caution is needed in the use of thermal or mechanical techniques on these sites because of the potential for damage to the soils resource. Another factor which can complicate tending young stands is the need to control species, such as sedges and lupine, which are desired feed for pocket gophers. In many situations, baiting and trapping of gophers alone will not limit conifer damage to acceptable levels. When fitted to the proper site conditions, however, nonchemical methods may provide satisfactory results.

The rationale for an increased effectiveness of herbicide use for site preparation or release in certain true fir-shrub-grass complexes is similar to that used in the ponderosa pine-grass model. In some situations, alternative methods will provide satisfactory results. In other circumstances, however, herbicides can more effectively control competing vegetation. These situations, described above, are a heavy grass or weed cover in proximity to crop trees, or where site variables limit the use of nonchemical methods.

As in the pine-grass analysis, broad scale, whole-area manual scalping of dense grass is considered operationally infeasible. Control of vegetation for the purpose of limiting rodent populations is also needed in some areas. On steep terrain, this can only be accomplished through the use of herbicides.

It is estimated, based on a survey of Forests, that loss of herbicide availability will be a significant factor on approximately 10 to 15 percent of the total acres of this vegetation type. For modeling purposes, the growth effect, due to loss of herbicide use on these sites, is considered similar to those under a "no vegetation management" scheme. Therefore, the potential long-term sustained yield is estimated to be reduced 7 percent (adjustment shown in Step 3 times 12-1/2 percent).

Step 5 Area Expansion

Information and Research Needs

In addition to the true fir-hemlock/shrub/herbaceous complex, there is little data and documented knowledge to quantify effects in several other extensive brush types. Two of the most prominent are the vine maple and ninebark influence areas. There is a need to better define the phenological characteristics and growth potentials of the vegetation, as well as their effects on conifer seedling development.

Vegetation management program research needs are well described by Walstad and Kuch (1987). Three phases of program improvement are identified. These are: Treatment Development and Evaluation; Treatment Decision Criteria; and Problem Prevention.

(a). Treatment Development and Evaluation

This aspect involves the development of alternative treatments for control of competing vegetation, the evaluation of treatment cost and effectiveness, and the identification of biological and physical factors affecting treatment effectiveness.

(b). Treatment Decision Criteria

Information needs include the relationship between stand growth and weed population density; evaluation of treatment timing and its influence on the necessary degree of control; incorporation of stand density influences on the level of competing vegetation effect in growth and yield simulators; and basic studies of tree and weed competitive interactions.

(c). Problem Prevention

This aspect offers the best long-term opportunities for minimization of weed problems. Included is the evaluation of complete reforestation and stand management systems for reduction of weed vegetation problems. Another important area of study is in weed ecology (especially ecesis—the establishment of a plant in a new habitat) and the role of early seral species in nutrient cycling, nutrient retention, and maintenance of long-term productivity.

Much work addressing these management needs is being conducted by agency, industry, and university organizations. Cooperative approaches with pooled resources are a likely trend because of the complexity of the problem and limitations of individual organizations.

Summary of Potential Timber Growth and Yield Effects

The following table (next page) summarizes timber yield effects, as they will eventually translate into changes in long-term sustained yield levels. A “no vegetation management” scheme is representative of program effects under Alternative C, while “no herbicide use” is a representation of Alternative A.

On a Regional basis, a loss of all vegetation management for young stand establishment in maintenance appears to trigger a 25 to 50 percent falldown in long-term yields, or approximately 1,000 to 2,000 million board feet annually.

Actual harvest level adjustments, if necessary, will be assessed by each individual Forest. Only that component of the capable-available land base managed for timber yields approaching the full biologi-

cal site potential will generally be affected. In many situations, the lack of vegetation management and/or herbicide use will limit the potential long-term sustained yield level achievable from a site. The relationship of the programmed harvest level (Allowable Sale Quantity) under a Forest Plan to this potential (long-term sustained yield) will determine the need for program adjustments.

Table A-1

Potential Long-Term Sustained Yield Reductions (Percent)

<i>Vegetation</i>	<i>No Vegetation Management</i>	<i>No Herbicide Use</i>	<i>"Full-Yield" Component* (1,000 Acres)</i>
Douglas-fir/Alder	-25%	(None)	236.8
Douglas-fir/Hemlock/Salmonberry	-21%	(None)	195.1
Ponderosa Pine/Grass-Forb	-52%	-4%	912.6
Douglas-fir/Ponderosa Pine/ Ceanothus spp.	-39%	-5%	1,436.2
Douglas-fir/Tanoak/Madrone	-65%	-19%	169.3
True Fir-Hemlock/Shrub/Herbaceous	-56%	-7%	1,474.7

* This represents 54.2 percent of the intensively managed suitable timberland base classified in the Forest land management process.

The suspension of herbicide use will reduce treatment effectiveness in those specific conditions identified. Regionwide, the effect on long-term yields appears to be a 2-1/2 to 3 percent falldown (based only on the reduced efficacy—cost effects are addressed in Chapter IV, Environmental Consequences). This represents an annual reduction of approximately 100 to 120 million board feet.

Subregional Effects on Timber Yields

Here, Regional long-term sustained yield (LTSY) effects are displayed for individual subregions. While the analysis of growth and yield (see Appendix A) is relatively comprehensive for the six vegetation complexes assessed, the reliability of numerical estimates decreases when disaggregated to smaller geographic areas. A rough estimate will be made, however, in order to characterize possible changes in timber yields over time for eight subregions:

<i>State</i>	<i>Subregion</i>	<i>Forests</i>
Washington	Coastal	Olympic
	Western Cascades	Gifford Pinchot, Mt. Baker- Snoqualmie
	Transition	Wenatchee
	East-side	Okanogan, Colville
Oregon	Coastal	Siuslaw, Siskiyou
	Western Cascades	Rogue River, Willamette, Umpqua, Mt. Hood
	Transition	Deschutes, Winema
	East-side	Ochoco, Wallowa-Whitman, Umatilla, Malheur, Fremont

Potential long-term sustained yield changes within subregions are presented in Chapter IV as a range of annual cubic foot volume adjustments.

The percentage adjustments calculated in this appendix have been expanded by the range of long-term sustained yield for alternative management strategies under consideration in the current Forest land management planning process. (Process records are on file at Forest Pest Management, Pacific Northwest Region, Portland, Oregon.) This has been done in order to:

1. link potential yield changes to the long-term sustained yield under consideration in the Forest Planning efforts;
2. provide an estimate of the possible magnitude of timber yield adjustments over time; and
3. emphasize that there are large differences in timberland productivity between the subregions.

Alternative A Process steps for allocating yield effects on a subregional level are:

1. Allocate yield effects to subregions by prorating acres in the four vegetation complexes in which yield falldown is anticipated, given the absence of herbicide use. (See Appendix A.) These complexes are:

<i>Complex</i>	<i>LTSY Change (%)</i>
Mixed Conifer/Tanoak/Madrone	-19
Mixed Conifer/Ceanothus spp./Herbaceous	- 5
True fir/Hemlock/Shrub/Herbaceous	- 7
Ponderosa Pine/Shrub/Herbaceous	- 4

2. Yield effects will be arbitrarily adjusted south and west within subregions to reflect the relative severity of moisture stress, intensity of site competition, and historic reliance upon herbicide use for maintenance of treatment effectiveness (rather than a cost-of-doing-business consideration). In general, a plus or minus 50 percent of the yield effects predicted has been applied.

Here is the relationship used to prorate the long-term sustained yield (LTSY) effect:

Change in LTSY (%) =

$$(A^* \text{ acres} \times \text{net yield}) + (B^* \text{ ac.} \times \text{net yield}) + (C^{**} \text{ ac.} \times 1.00)$$

$$(\text{Total acres programmed for "full yields"}^{***})$$

* A, B = Vegetation complex in which yield reductions will occur.

** C = Vegetation complex in which no yield falldown is shown.

*** D = Number of acres producing full potential timber yield.

Washington

<i>Subregion</i>	<i>Acres Affected</i>
Coastal:	0 ac. affected
Transition:	0 ac. affected
East-side:	0 ac. affected
Western Cascades:	27 m ac. @ -5% (ceanothms)
	262 m ac. @ -7% (true fir)

$$\text{LTSY (\%)} = \frac{(27 \text{ M} \times .95) + (262 \text{ M} \times .93) + (431 \text{ M} \times 1.00)}{720 \text{ M}^*} = .973, \text{ or minus } 2.7\%$$

*M = 1,000

Oregon

Coastal: 106 M ac. @ -19% (tanoak)
 44 M ac. @ -5% (ceanothus)
 27 M ac. @ -7% (true fir)

$$\text{LTSY (\%)} = \frac{(106 \text{ M} \times .81) + (44 \text{ M} \times .95) + (27 \text{ M} \times .93) + (388 \text{ M} \times 1.00)}{565 \text{ M}^*} = .957 \text{ or minus } 4.3\%$$

Westside Cascade: 532 M ac. @ -5% (ceanothus)
 219 M ac. @ -7% (true fir)
 23 M ac. @ -19% (tanoak)

$$\text{LTSY (\%)} = \frac{(532 \text{ M} \times .95) + (219 \text{ M} \times .93) + (23 \text{ M} \times .81) + (1,040 \text{ M} \times 1.00)}{1,814 \text{ M}} = .974 \text{ or minus } 2.6\%$$

Transition: 630 M ac. @ -4% (tanoak)
 110 M ac. @ -5% (ceanothus)
 255 M ac. @ -7% (true fir)

Eastside: 807 M ac. @ -4% (ponderosa pine)
 711 M ac. @ -7% (true fir)
 205 M ac. @ -5% (ceanothus)

$$\text{LTSY (\%)} = \frac{(807 \text{ M} \times .96) + (711 \text{ M} \times .93) + (205 \text{ M} \times .95) + (390 \text{ M} \times 1.00)}{2,113 \text{ M}} = .956 \text{ or minus } 4.4\%$$

Following the adjustments referred to in step 2, the estimated subregional change in long-term sustained yields are:

<i>Subregion</i>	Washington	Oregon
Coastal*:	No Change	Minus 5-8%
Transition:	No Change	Minus 2-3%
East-side:	No Change	Minus 2-3%
Western Cascades*:	Minus 1/2%	Minus 2-4%

*Note that yield effects would be concentrated on Forests within the southern portion of these subregions.
 M = 1,000

This alternative is used as a reference for evaluation of yield effects. Forests will realize the full Forest Land Management Plan Allowable Sale Quantity (ASQ) and Long-Term Sustained Yield (LTSY) under the implementation of Alternative B.

Alternative B

Estimates of yield reductions in the complete absence of vegetation management are made for the six vegetation complexes addressed in Appendix A. Extrapolation must be used to characterize yield fall-down for forestlands not represented in the detailed analysis. Despite the broad numerical ranges indicated in the growth and yield analysis, it is apparent that implementation of Alternative C will result in catastrophic reductions in timber yield over time because of high levels of tree mortality, growth suppression, and vigor or form damage.

Alternative C

An approximation of potential LTSY changes by subregion is presented:

Potential LTSY Reduction	Subregion
Less than 20%	Coastal (WA), Western Cascades (WA) Transition (WA), East-side (WA)
20 to 40%	Transition (OR), East-side (OR)
Greater than 40%	Coastal (OR), Western Cascades (OR)

An increased proportion of the silvicultural site preparation and release diagnosis will result in a no-treatment or deferred-treatment decision under Alternative D in comparison with Alternative B.

Alternative D

No Treatment Decision (thousands of acres)

	<i>Alternative B</i>	<i>Alternative D</i>
Site Preparation	8	17
Conifer Release	3	9
	11	26

The 15,000 acre reduction in program accomplishment is a response to the added caution, reduced tolerance for uncertainty of vegetative response, and emphasis on preventive rather than corrective action. While all Forests would be affected under this management emphasis, the bulk of program reduction would occur on Oregon National Forests. Over time, the reduced work accomplishment is expected to translate in a 1-1/2 to 2% change in long-term sustained yields.

An apportionment to subregions is based on factors such as the relative severity of competing vegetation and moisture stress, and the level of uncertainty regarding conifer growth and yield effects from dominant competing hardwood, shrub, or herbaceous species.

A crude approximation of timber yield effects on individual subregions is presented below.

<i>LTSY Change</i>	<i>Subregion</i>
Minus 2 to 3%	Coastal (OR), Western Cascades (OR)
Minus 1 to 2%	East-side (OR), Transition (OR)
Less than 1%	Western Cascades (WA), Coastal (WA), East-side (WA), Transition (WA)

It must be emphasized that this disaggregation from Regional to subregional estimates is simply to characterize relative differences in the magnitude of effects. Actual local effects must be addressed by each National Forest within the context of management plan direction, and timber management program priorities and opportunities.

Alternative E

A reduced effectiveness in site preparation and release efforts is anticipated on Forests which have had historic aerial herbicide application programs, or which rely heavily on manul (chainsaw) release. In general, this includes Forests within the Coastal (OR) and southern portion of the Western Cascade (OR) subregions. A combination of relatively rugged terrain and severe brush or hardwood competition is characteristic of these areas. A less important feature of the alternative is the slightly larger (1,000 acres annually) no-treatment/deferred treatment conifer release diagnosis in comparison with Alternative B.

Presented below is a rough approximation of subregional potential changes in long-term sustained yield:

<i>LTSY Reduction</i>	<i>Subregion</i>
Minus 3 to 5%	Coastal (OR)
Minus 1 to 3%	Western Cascades (OR)
Little or no change	Coastal (WA), Transition (WA), Western Cascades (WA), East-side (WA), Transition (OR), East-side (OR)

Alternative F

The loss of burning as a site preparation technique will be most damaging for Forests which have heavy volumes of competing vegetation (highly productive sites) and steep terrain in combination. This mix of factors can lead to understocked inclusions in managed stand, jackpots of debris in depressions, and some areas which are simply physically unplantable due to obstructions following timber harvest. Also affected will be Forests which use broadcast burning for seedbed preparation in an effort to encourage natural regeneration.

Subregional effects will tend to be greatest in the Coastal (both Oregon and Washington) and Western Cascades (OR). An estimation of long-term sustained yield reduction is:

<i>Minus 3-5%</i>	<i>Minus 1-3%</i>	<i>Little or No Change</i>
Coastal (WA)	Western Cascades (WA)	East-side (WA)
Coastal (OR)	Transition (WA)	Transition (OR)
Western Cascades (OR)	East-side (OR)	

The enlarged program of work under this alternative will increase site preparation and release efforts by 23 percent annually.

Alternative G

Total Yearly Cultural Program (thousands of acres)

<i>Method</i>	<i>Alternative B</i>	<i>Alternative G</i>
Manual	11	13
Chemical	29	42
Mechanical	19	20
Biological	3	4
Thermal	18	22
No-Treatment	11	11
Total	92	112

An improved stocking level in some difficult reforestation conditions is the principal silvicultural benefit seen under this alternative. This appendix identifies three vegetation complexes where seedling mortality is particularly severe in the absence of vegetation management. These are:

1. Douglas-fir/Tanoak/Madrone
2. Ponderosa pine/Shrub/Herbaceous
3. True-fir/Hemlock/Shrub/Herbaceous

All Forests appear to have opportunities to benefit from a more aggressive site preparation and release program. The greatest potential for improved yields, however, will be in those Forests with a large proportion of the three identified vegetative conditions. The approximation of subregional changes in LTSY is based on this premise. The following list indicates those Forests with a portion of the "Full-Yield" timberland component in high potential seedling mortality vegetative conditions.

<i>Forests</i>	<i>Affected Ac. (M)</i>	<i>Proportion (%)</i>
Ochoco	280	85
Malheur	699	85
Wallowa-Whitman	126	83
Deschutes	665	70
Mt. Baker-Snoqualmie	177	60
Fremont	315	55
Umatilla	98	41
Winema	165	38
Siskiyou	133	38
Rogue River	77	28
Mt. Hood	62	20
Gifford Pinchot	85	20
Willamette	73	10
Umpqua	30	6
Remaining Forests (5)	0	0

As with Alternative A, an arbitrary shift of effects will be made to the south and west within subregions. This is done in order to approximate the relative change in moisture stress and severity of competition which is seen in each of the three vegetation complexes.

An approximation of subregional potential increases in long-term sustained timber yields is made as follows:

<i>LTSY Increase</i>	<i>Subregion</i>
3 to 5+%	East-side (OR), Transition (OR)
1 to 3% *	Coastal (OR), Western Cascades (OR)
	Western Cascades (WA)
Less than 1%	Coastal (WA), Transition (WA),
	East-side (WA)

* Potential effects are greatest in the southern portion of the subregion.

It must be emphasized again that these are simply estimates shown in order to characterize the relative change in yield effects throughout the Region. Large potential error terms are associated with the numerical estimates in this type of disaggregation. Each individual National Forest would evaluate opportunities for improved stand regeneration and sound stand maintenance during the actual implementation of Alternative G.

Appendix B

Economic Efficiency Analysis

B

Appendix B

Economic Efficiency Analysis

This section describes the costs and benefits (as well as some concepts involved in economic efficiency analysis), how they were derived, and how they were used in developing the Region's vegetation management program.

Descriptions of Some Concepts Related to the Efficiency Analysis

Priced Outputs (Benefits)

Priced outputs are those that can be exchanged in the market place. Their quantitative values are determined by actual market transactions or by estimation methods that produce prices approximately those determined by market transactions.

Timber and forage are examples of commodities that are bought and sold in the market. Their values are determined through the interaction of buyers and sellers, based on supply and demand conditions in the market at the time of the transaction. Recreation visitor days (RVD's), on the other hand, are not normally exchanged via market transactions. Their market values are estimated by using some market transaction data in combination with various theoretical techniques.

Conceptually, these assigned values are consistent and comparable to those values which were actually derived via market transactions (Rosenthal and others 1985). Therefore, both assigned and market values are appropriate for calculating quantitative measures of efficiency such as present net value. All benefits and costs are tracked in real terms, i.e., exclusive of inflationary effects.

Nonpriced Outputs

Nonpriced outputs are those for which there is no available market transaction evidence, and no reasonable basis for estimating a dollar

value comparable to market values associated with the priced outputs.

In these cases, subjective nondollar values must be attributed. These values are therefore described qualitatively rather than quantitatively. They may be either positive or negative. In fact, what may be considered to be a benefit to one individual may represent a cost to someone else.

Examples of nonpriced outputs include threatened and endangered species, natural and scientific areas, historical and anthropological sites, visual quality, and clean air.

Discounting

Analyses of alternative investment options usually involve cash flows over different periods of time. Due to a number of factors, including the uses to which it can be applied, a dollar received today is worth more than a dollar received ten years from now. Discounting is a process whereby the dollar values of costs and benefits which occur at different periods are adjusted to a common time period so that they can be compared.

Usually the common time period is the present, in which case the discounted value is referred to as a present value. The real discount rate used in the analysis is 4 percent. As a real discount rate, it is exclusive of the effects of inflation. The 4 percent real discount rate was also used in Forest planning. A 7-1/8 percent discount rates is used to show the sensitivity of the model to the selection of a particular real discount rate.

Present Net Value (PNV)

Present net value is the difference between the discounted value of all outputs or benefits for which monetary values or established prices are assigned and the total discounted dollar costs of managing the planning area.

The period of analysis used is 100 years. This enables the model to reflect the changes that occur as the Forests are changed to a managed condition. Costs and benefits beyond that point are reduced to insignificance through discounting. Present net value calculations consider only the benefits and costs for which dollar values were assigned.

Priced benefits included timber and domestic livestock grazing. These benefits were compared with their related costs. PNV is an estimate of the respective alternative's excess of dollar-quantified benefits over its related dollar-quantified costs.

Opportunity Costs

Opportunity costs are defined as the value of a resource's foregone net benefit in its most economically efficient alternative use (FSM 1970.5).

In relation to the economic analysis performed for this analysis, it represents the decrease in PNV which occurs when an alternative other than the one with the highest level of PNV is selected. Therefore, opportunity costs measure the net change in PNV for priced resource outputs and associated costs, and can be used to measure the net value sacrificed in order to produce the nonpriced benefits included in net public benefits.

Net Public Benefits (NPB)

Maximization of net public benefits is the goal of the analysis process. Net public benefits is the overall value to the Nation of all outputs and positive effects (benefits) less all the associated inputs and negative effects (costs) whether they can be quantitatively valued or not. Net public benefits cannot be expressed numerically because the concept includes qualitatively-valued nonpriced outputs.

Conceptually, net public benefits is the sum of the present net value of priced outputs plus the net value of all nonpriced outputs.

Welfare Distribution Effects and Impacts

There is another level of effects which are also a concern to management. These are the welfare distribution effects influenced by the mix and level of outputs produced by the National Forests. They can be either positive or negative. They can be local, regional, or national in scope.

Some distributive effects such as changes in consumer prices or taxpayer costs have national level impacts. Others, such as induced jobs and income or payments to local governments, are more local or regional in nature. These concerns are more related to questions of equity (i.e., who pays and who benefits) rather than questions of efficiency. They are not assessed in the context of the efficiency criteria associated with PNV and net public benefits. However, these positive and negative distributive effects need to be assessed as part of the net public benefit measures, since equity objectives often influence efficiency objectives and vice versa.

In order to calculate the present net value for each alternative, assumptions had to be made regarding discount rates, demand functions, establishing a common reference point in time for dollar values, and real price and cost trends. This section summarizes these decisions and their resulting parameters.

Discount Rates

Discounting involves the application of a discount rate that represents

Parameters and Assumptions Used For Economic Efficiency Analyses

the time value of money in determining the present value of future costs and benefits. A 4 percent real discount rate was used to calculate the present net value for each benchmark and each alternative.

As a real discount rate, it excludes the effects of inflation. This is not an assumption that there will be no inflation. It is rather an assumption that the costs and benefits presented in the analysis will generally increase or decrease at the same rate as the rate of inflation. According to FSM 1971.71:

“For evaluations of long-term investments and operations in land and resource management in the 1980-1985 planning period, a 4 percent real discount rate shall be used. Evaluations should also discount benefits and costs at the real discount rate used in the most recent RPA to determine sensitivity of alternatives to variations in the discount rate.”

The 4 percent rate approximates the “real” return on corporate long-range investments above the rate of inflation (Row and others 1981). The 4 percent rate was used to run FORPLAN during Forest planning. The 1985 RPA Program used a real discount rate of 7-1/8 percent. An analysis of the sensitivity of the preferred alternative to the discount rate was performed using the 7-1/8 percent discount rate as well. All costs and benefits are discounted from the midpoint of the decade in which they are expected to be incurred.

Demand Functions and Real Price Trends

As specified by the Washington Office, USDA Forest Service (1920 letter to the Regional Forester, “Downward Sloping Demand Curves,” 2/3/81), and in keeping with FSM 1971.65, horizontal demand functions for timber and nontimber resources were used to analyze the alternatives for the DEIS. This was also consistent with the methods used in the ongoing Forest planning process. Many factors can influence the demand for stumpage from any one Forest (Row and others 1981).

Some of these factors include trends in interest rates; the species and product mix; the use of wood for energy; forest products exports; the cost of Canadian lumber; the rate of technical improvement in wood processing; and the levels of harvests on other National Forests. Also to be considered are the full range of wood product substitutes and the full range of considerations dealing with their respective, complementary goods and practices.

All these contain some degree of uncertainty. Neither the empirical nor the theoretical bases have been well enough developed to derive reasonable estimates of the demand functions for the resources offered at the Forest level. Evidence suggests that the elasticity

in the portion of the timber demand curve for which the Forest can influence output levels is such that prices would be relatively insensitive to some "reasonable" range of Forest timber offerings.

In other words, it appears that the timber demand curve for the range of output levels analyzed during the development of alternatives is best depicted by a horizontal demand function. We assume that prices are insensitive to volumes offered. The assumption is admittedly strained in the case of Alternative C. The resulting figures for Alternative C should nevertheless provide a good basis for comparing it to the other alternatives.

Real price trends were developed and used to represent the rate at which resource values will change over time as a result of anticipated supply and demand interactions in the market place. As specified by the Regional Office (1920 letter to Forest Supervisors, "Timber Price Trends, Values, and Costs", 9/25/84), a 1 percent per year increasing real price trend for stumpage was used for FORPLAN harvest scheduling analyses. This was applied for the first 50 years. No further change in the price trend was assumed for the remaining 50 years of the planning horizon.

Nominal stumpage prices (i.e., those which include the effects of inflation) are therefore increased during the next 50 years at a rate of 1 percent greater than the rate of inflation. Thereafter they are assumed to change at a rate equal to the rate of inflation. Use of this assumption helps maintain consistency with the Forest planning process currently underway in the Region and does not serve to skew results.

Consistent with Washington Office direction used in the ongoing Forest planning process, constant real prices were assumed for all other resources.

Real Cost Trends

Based on Washington Office direction used in the development of the Region's Forest Plans, constant real costs were assumed. That is, the costs of labor, fuels, materials, and all other factors of production involved with managing the Forest were assumed to change at a rate equal to the rate of inflation.

Real Dollar Adjustments

All costs and values used in the Forest planning process are expressed in 1987 dollars. The GNP Implicit Price Deflator was used to convert historical nominal prices and costs to this common base (FSM 1971.32b). Because the Deflator had not yet been estimated for 1987 at the time of the analysis, the rate of increase for the period 1982 through 1986 was assumed to continue for the additional year.

Externalized Costs and Benefits

Because of Forest Service management, benefits and costs accrue to others. These externalized costs and benefits are not tracked in the economic efficiency analysis. Insofar as we have been able to identify them as significant, varying by vegetation management alternative, and insofar as we have been able to quantify them, we have recorded them in the appropriate sections of these documents.

For instance, as a result of Forest Service management, certain jobs associated with livestock management exist. We identified those jobs (as well as personal income and payments to local governments) in the local economic impact analysis. Other externalized costs and benefits relate more directly to concerns of economic efficiency—such as the spread of noxious weeds or wildfire from public to private land.

Such concerns are real, but they have not been addressed quantitatively in the economic efficiency analysis. The decisionmaker must address those and similar concerns qualitatively in arriving at a decision as to which alternative it is that best maximizes net public benefits.

Internal (to the decision) effects such as the effects on the allowable sale quantity are included in the analysis to the extent that they have been identified as being significant and significantly different by alternative.

Costs Used for Economic Efficiency Analyses

This section describes the costs used to perform economic efficiency analysis for each of the alternatives considered during the development of the DEIS.

All Forest Service costs relevant to the question of vegetation practices were included for purposes of estimating budgets and calculating the present net value for each alternative. These costs were identified by their 1989 outyear budget codes. The outyear budget codes with their related descriptions were useful for identifying how different costs would be treated during the planning process. Because each Forest was solicited for cost and benefit data, the use of this common reference point was needed to maintain comparability. All participants had the same definitions—the same common denominator.

During the course of the analysis it became apparent very early that there would be many Forest Service costs which would not be affected at all by the analysis currently underway. They simply were not affected by vegetation management processes. It was therefore not necessary to include those costs in the analysis. And of course their related benefits were also excluded.

It is important to remember, though, that because the analysis

did indicate that Forest outputs such as timber might be affected by the choice of alternative, that the costs and benefits which vary with timber sale offerings were included in the analysis.

The same situation applies to permitted livestock grazing. Additionally, some costs incurred by the public in their use of the National Forests were tracked.

Fixed Costs

A cost was classified as being “fixed” if it was not expected to vary significantly over the range of alternatives considered. Fixed costs were a component of the budget estimates so that the decisionmaker could view prospective changes in the light of the overall budget. Because they are fixed, they do not vary among the alternatives presented.

Table B-1 (next page) lists the 1989 outyear budget codes used in the analysis to track how the alternatives affected dollar expenditures in individual line items. Other budget codes were addressed in aggregate to reflect changes which occurred when vegetation management practices affected Forest outputs, viz., timber and livestock grazing.

Variable Costs

All other costs are classified as being “variable”. For instance, individual Forests have the ability to invest more or less money in vegetation management practices in response to the overall theme of an alternative. They also have the ability to change the levels of their outputs as the situation indicates.

HERB, an electronic spreadsheet representing all quantified costs and benefits, was used to generate final figures for presentation.

Budget Considerations

During the analysis, cost data was solicited from every National Forest in the Pacific Northwest Region as well as from other sources.

Table B-2 shows estimated budget impacts resulting from implementation of the various vegetation management alternatives. The information is presented in this way so that the decisionmaker as well as other affected parties can better see the effects on individual programs and line items. Presented in aggregate, significant changes might be masked. The table shows the effects on various forms of vegetation management.

Table B-1

1989 Outyear Budget Costs Directly Affected by Vegetation Management Practices

Code	Description
AT23	Trail Maintenance
CW222	Wildlife Habitat Nonstructural Improvements
CT222	Threatened and Endangered Species, Nonstructural Habitat Improvement
DN222	Range Resource Nonstructural Improvement
DN24	Noxious Farm Weeds
ET241	Reforestation—Site Preparation
ET251	Timber Stand Improvement—Release and Weeding
ET252	Timber Stand Improvement—Precommercial Thinning
ET27	Genetic Tree Improvements
JL231	Landline Maintenance
LF23	Facility Maintenance
LT23	Road Maintenance
PF1	Fire Management Operations
PF12	Fire Suppression
PF24	Natural Fuels Improvements
PF25	Activity Fuels Improvements
	Activity Fuels Improvements—Purchaser
ET113 (NFAF)	Timber Resource Coordination
FA	Air Resource Activities

A review of Table B-2 provides a nutshell view of how different vegetation management activities would be accomplished under the different alternatives. Fire suppression costs, for instance, generally run contrary to the intensity of slash management in any alternative. Genetic Tree Improvement costs generally reflect the degree of difficulty expected in managing vegetation in any alternative. Range Resource Nonstructural Improvements reflect in their own particular way the problems associated with trying to identify substitute methods that can be cost-effective.

Benefits Considered in Economic Efficiency

This section describes the benefits which were incorporated in the economic efficiency analyses for each alternative considered during the development of the DEIS.

Resource outputs for which dollar values were assigned consti-

Table B-2

Direct Budget Effects Resulting From Vegetation Management¹

(Figures Shown Are Relative to Alternative B, a Likely Regional Level)

(Average Annual Difference over the Next Ten Years, 1987 Thousand \$)

Trail Maintenance	0	REF.	-336	-177	-8	+7	+20
Wildlife Habitat Nonstructural Improvements	0	REF.	-1,106	-62	-123	-274	+160
Threatened and Endangered Species, Nonstructural Habitat Improvement	0	REF.	-58	0	0	0	0
Range Resource Nonstructural Improvement	-188	REF.	-368	-135	0	0	-134
Noxious Farm Weeds	-27	REF.	-302	-31	+4	+5	+474
Reforestation—Site Preparation	+930	REF.	-8,879	-1,291	+347	-707	+1,025
Timber Stand Improvement—Release & Weeding	+2,382	REF.	-4,409	+669	+1,312	+609	+6,923
Timber Stand Improvement— Precommercial Thinning ²	+662	REF.	-12,056	-2,320	-339	+91	+646
Genetic Tree Improvements	+389	REF.	-1,019	+112	+12	+485	+83
Landline Maintenance	+7	REF.	-41	-24	+18	+18	+21
Facility Maintenance	+123	REF.	+13	-102	+99	+99	+5
Road Maintenance	+1,600	REF.	-139	+69	-69	-17	+413
Fire Management Operations	0	REF.	+3,215	+581	0	+557	-1,012
Fire Suppression ³	0	REF.	+26,430	+8,321	+3,486	+5,281	-345
Natural Fuels Improvements	0	REF.	-1,463	-246	-13	-66	+538
Activity Fuels Improvements	+246	REF.	-20,796	-7,428	-5,259	-8,849	+2,090
Activity Fuels Improvements' Purchaser ⁴	+58	REF.	-16,669	-7,482	-2,345	-5,132	+297
Timber Resource Coordination ⁵	-7	REF.	-474	-102	-45	-34	+43
Air Resource Activities	0	REF.	-18	-14	0	-6	+72

REF. (reference) indicates the situation expected with implementation of the Forest Plans currently being developed.

¹ Vegetation management is done in the pursuit of other goals or objectives, such as increasing timber production or providing for public safety along roadways. The costs shown in this table reflect only the direct costs associated with vegetation management in the work areas shown. Variation in vegetation management practices causes changes in other program work areas – causing changes in their associated costs. Those costs are not included in this table.

² Slash treatment component only.

³ Fire Suppression costs are tracked after the fact—they are not included in budget proposals. They are shown in this table to provide the decision maker with a more complete view.

⁴ These are not budget costs. These are costs incurred by the timber purchaser. They result in lower bids on Forest stumpage.

⁵ These are costs incurred by the Protection budget element in support of the timber program.

tute the benefits included in the present net value calculations. Like the costs included in the analysis, only those benefits incurred during the 100-year planning horizon were incorporated in the PNV calculations. This period of analysis is adequate to capture all significant residual costs and benefits.

Benefits Whose Values Are Determined in the Marketplace

Benefits fall into one of two categories: market or non-market (assigned). Market values constitute the unit price of an output normally exchanged in a market after at least one stage of production, and are expressed in terms of what people are willing to pay—as evidenced by market transactions.

Nonmarket values constitute the unit price of an output not normally exchanged in a market at any stage before consumption, and thus one whose value must be assigned from other information (FSM 1970.5). They are valued in terms of what a reasonable and prudent individual would be willing to pay (above participation costs) rather than to go without.

In either case, their values are theoretically commensurate and appropriate for inclusion in PNV calculations. The resources for which dollar values were estimated in this analysis were timber and domestic livestock grazing. These were the only dollar-quantified outputs which were found to vary significantly among the alternatives. As FSM 1971.63—1 puts it:

“Values should only be determined for outputs that are sold or could potentially be sold if the law or Forest Service policy permitted.”

Values used for timber (stumpage) have been adjusted to exclude those sales which were sold at a premium but were never harvested. The values are representative of a six-year period to help compensate for the unevenness of demand for timber over time.

The range outputs represent the amounts of forage to be grazed by domestic livestock and are measured in animal unit months (AUM's). AUM values represent permittee willingness-to-pay for grazing on the respective National Forests. The Forest Service entered into a cooperative agreement with the USDA Economic Research Service to develop livestock enterprise budgets for each National Forest. The Range Budget Approach was used for this analysis. Because Forest AUM's are not actually priced in a free competitive market, the calculated price is an estimate of market value. The Regional Office Direction Package of April 27, 1984 identifies the Forest-respective values.

This is not to say that individuals will not be inconvenienced

by vegetation management practices (including “no action”). Rather, it is to say that such practices normally are done during a short period of time; that they are timed so as to cause the least possible inconvenience to recreationists; and that their effects per se are generally regarded as being transitory in nature.

Some recreation may be lost due to the form of vegetation management specified in the final preferred alternative, but some may be gained as well.

Because recreational use of the National Forests was not expected to be significantly affected by implementation of any of the alternatives under consideration, it was not included in the analysis. Other National Forest programs were included in the analysis insofar as their costs were affected by the alternatives under consideration.

Consideration of Nonquantified Costs and Benefits

The calculation of PNV provides efficiency data to use when comparing alternatives. However, other factors also influence the decision-making process. In some cases the importance of nonpriced benefits, for which it is unrealistic to assign monetary values, can outweigh the advantages of producing a higher level of PNV.

The importance of the need to consider these subjectively-valued benefits in Forest management decisionmaking is addressed in the NFMA Regulations which charge the Forest Service with identifying the alternative which comes nearest to maximizing net public benefits (36 CFR 219.12(f)).

Net public benefits (NPB) represent the overall value to the nation of all outputs and positive effects (benefits) less all associated inputs and negative effects (costs), whether they can be quantitatively valued or not (36 CFR 219.3).

Net public benefits include both priced and nonpriced resource outputs, less all costs associated with management. As stated earlier, all priced outputs and costs associated with vegetation management on the National Forests are included in the calculation of PNV. To this, the net subjective values of the nonpriced outputs must be included in order to arrive at the overall NPB of an alternative. Some of the more important nonpriced concerns addressed during the planning process included the following:

Public Health

Public Participation

Social and Economic Effects

Environmental Effects

Effectiveness of Techniques

Interagency Coordination

These are outputs and effects which are influenced by Forest management. They are all related to one or more issues and concerns which were identified at the outset of the planning process.

The nonpriced outputs considered during the development and evaluation of alternatives are discussed below. While the quantitative dollar values of each are not determined, they can generally be evaluated by examining such quantitative indicators as acres of treatment.

Public Health: Health issues related to the management of vegetation have been a major focus during the past decade and continue to be a concern. Much attention centered on the safety of herbicides used in vegetation control. We received many comments on the need to evaluate human health impacts. Management is attuned to these concerns just as they are to the numerous other concerns frequently voiced regarding management of the National Forests. Public perception of health risks are very real concerns—whether the scientific literature supports them or not.

Public Participation: The extent to which affected parties feel they have a say in the decisionmaking process affects the degree to which they will participate in the process. It also affects their feelings of independence and self control.

Social and Economic Effects: Vegetation management activities have both direct and indirect effects on employment, personal income, payments to local governments, and the overall quality of community life. Many of these effects have been quantified but their weight in the overall consideration of net public benefits is left to the decisionmaker.

Environmental Effects: There is widespread concern about the physical and biological effects on the environment of using vegetation management techniques. The decisionmaker will need to consider the complex physical and biological linkages; the sensitivity of ecosystems; and the direct, indirect, long-term, and cumulative effects.

Effectiveness of Techniques: Vegetation management techniques vary in their effectiveness depending on site-specific conditions. This variability in effectiveness is better documented for some practices than for others.

Interagency Coordination: Agencies at all levels of government have a shared interest in resource management through vegetation manipulation. Many will be directly affected by decisions made in the EIS, others indirectly. Many agencies reminded us of cooperative agreements, administrative or cost impacts, and shared legal responsibilities or liabilities—all factors of the decisionmaker to consider.

Social and Economic Impact Analysis

Area of Influence Established for Economic Analysis

To assess the current economic conditions and to estimate potential changes, an Area of Influence was determined. This is the geographic area wherein the majority of Forest products are first used and wherein public concern is concentrated.

Social and Economic Overview

Table B-3
Area Population Over Time

	1940	1950	1960	1970	1980	1986
Oregon	1,090,000	1,521,000	1,769,000	2,091,000	2,633,000	2,660,000
Washington	1,736,000	2,379,000	2,853,000	3,409,000	4,130,000	4,420,000
Total	2,826,000	3,900,000	4,622,000	5,500,000	6,763,000	7,080,000

Source of 1940-1980 data: the decennial census. 1986 Oregon data calculated from Table 3 of Population Estimates of Oregon, Counties and Cities, July 1, 1986, published by the Center of Population Research and Census, School of Urban and Public Affairs, Portland State University. 1986 Washington data calculated from Table 9 of 1986 Population Trends for Washington State, August, 1986, published by the Office of Financial Management of the State of Washington.

Social Effects

Social effects are changes in communities, peoples’ beliefs, and their ways of life. Specifically, the social effects of the vegetation management alternatives are the different social conditions they cause.

Estimation of social effects is a two-step process. First, the conditions that are expected under the current management direction over the next 10 years are estimated. Then the factors that would change because of different management direction (alternatives) are noted and compared to the current management direction—or “baseline”—conditions.

The following categories encompass the significant social effects of the alternatives developed for vegetation management. The social effects for the Region are reported together and special note made where the effects on any subarea are substantially different.

Lifestyle and Job Dependence: Effects are indicated by significant changes from baseline conditions in the patterns of work, leisure, and other activities. Dimensions include the availability and characteristics of jobs, recreational activities, aesthetic and amenity ties to the National Forests, and the gathering and use of food and fuel from the National Forests.

Determination of Social Effects

Beliefs and Perceptions: Significant effects are indicated by outputs and practices at odds with the understandings and emotional values people have for the Forest.

Dimensions include concerns that familiar places will be changed; that full use of Forest commodities will be restricted; and that disliked or unsound practices will occur. People have concerns for visual qualities; for the Forests as a natural or managed ecosystem; for environmental qualities; and for historical and cultural sites and characteristics.

Sense of Control/Sense of Self-sufficiency: These effects are indicated by changes from the baseline conditions in the degree of control affected groups perceive they have over the National Forests compared to outside forces (e.g., government, interest groups, or industries). It also reflects the affected groups' sense that they can contribute to their subsistence needs (food, fuel, shelter) by their own direct knowledge and effort. Dimensions include concerns about government restrictions on uses and practices, and changes caused by special interest groups.

People who gain food or fuel directly from the National Forests are concerned about reduced access or supply. Because local area inhabitants live so close to the National Forests, their sense of control or self-sufficiency is more profoundly impacted by National Forest actions than are others. Next in order of significance would be the recreationists from outside the area who have, in many cases, been returning to the area for years. This latter group and other recreationists would suffer a loss in their sense of control or self sufficiency if their existing recreational enjoyment were to be reduced.

Categories of Social Effects Considered, But Not in Detail, for Each Alternative

In addition to the three main categories of social effects, the effects of the alternatives on population change and the effects on land use and ownership were examined.

Population Change: Selection of a vegetation management alternative may affect the level of economic activity in the Pacific Northwest Region. This may ultimately affect migration and population. Many short-term changes can, however, be accommodated without changes in population.

In recent years, migration has been responsive both to employment opportunities and to a variety of amenity and lifestyle factors so that a precise population projection tied to the vegetation management alternatives is not presented. However, employment and population are generally recognized as being directly related.

Landownership and Use Changes: Land exchanges will be primarily with private parties, corporations, or other government agencies which normally use the land for purposes similar to current National Forest management. Land for development is not significantly affected, either directly or indirectly, by the alternatives presented.

Input-Output Models (Alward and others, n.d.; Palmer 1976)

Any developed economy, whether national, regional, or local, is characterized by a high degree of interdependence among its producing sectors. Each economic sector not only produces goods or services but is also a consumer itself, purchasing other goods and services for use in the production process. Interindustry relations have been recognized for a long time. Francois Quesnay's "Tableau Economique" of 1758 developed the circular flow and general equilibrium concepts, while Walras stressed the interdependence between the production sectors of an economy with his general equilibrium model in the 1870's.

The first empirical application of the input-output model in the Anglo-American world dates from 1936 when Leontief published an input-output system of the United States economy.

What Leontief did was to simplify Walras' generalized model so the model's equation could be estimated empirically. He used two simplifying assumptions. First, the large number of commodities in the Walrasian model was aggregated into relatively few outputs, one for each industrial sector. Second, the supply equation for labor and demand equations for final consumption was abandoned and the remaining production equations were expressed in their simplest, linear form.

These simplifying assumptions make a sharp difference between input-output and most other conventional economic models. The linearity does not allow factor substitution or economies of scale. Time is missing, yet the purchase of inputs by one industry to make goods to sell to other industries implies a period analysis. The prevalence of joint products and multi-product plants makes it impossible to aggregate only those plants with similar output and input structures together.

However, the model is starkly simple. Its key variables are the outputs of sectors into which the economy is divided. Each sector's output consists of summing its sales to all other sectors and to final

**Use of the
IMPLAN
Computer Model*
to Estimate
Effects on Jobs
and Personal
Income in the
Local Area**

**A more recent version of IMPLAN is in the final proofing stage. It was therefore not used in this analysis.*

demand. The amount of each product which each sector consumes depends only on the level of output in the consuming sector. Equilibrium in the economy is attained when the output of each sector equals total purchases from that sector, these purchases being determined by the output of all other sectors.

Because of these simplifying assumptions and the simplicity of the model, it is empirically implementable. The implausible assumptions of the production function do not appear to restrict the model too badly. Technology changes are slow enough so the input coefficient matrix of one year seems to be good for several years. Even out-of-date tables can show the maximum input requirement. Perhaps most important of all, input-output models pass the critical test that for many purposes they predict reasonably well.

Development of IMPLAN

The USDA Forest Service developed IMPLAN to assist its land and resource management planning efforts in economic impact assessment. IMPLAN utilizes input-output analysis procedures to provide forest planners with the capability to develop nonsurvey-based inter-industry models and apply them to the evaluation of alternative management programs.

The IMPLAN data base consists of two major parts: (1) a national-level technology matrix and (2) estimates of sectoral activity for final demand, final payments, gross output and employment for each county. The data represent 1977 county level economic activity for 466 sectors as shown in Table B-4.

Table B-4

Contents of the IMPLAN Data Base for Each U.S. County

A. Final Demand

1. Personal Consumption Expenditures
2. Capital Formation
3. Inventory Change
4. State and Local Government Expenditures
5. Federal Government Expenditures
6. Foreign Exports

B. Final Payments

1. Employee Compensation
2. Indirect Business Taxes
3. Property-Type Income

C. Total Gross Output

D. Production Employment

The national technology matrix denotes sectoral production functions and is utilized to estimate local purchases and sales. This 466-sector, gross domestic based model was derived from the Commerce Department's 1972 national input-output model (U.S. Department of Commerce 1979a).

The "use" and "make" tables were rectified to an "industry by industry" basis and updated to 1977 using relative price changes and the RAS* procedure (used to update technical coefficients—Stone and others 1962) with the 1977 National Income and Products Accounts (U.S. Department of Commerce 1977a) information used as control totals. Aggregation of some agriculture, construction, and manufacturing sectors, and disaggregation of the mining sectors resulted in the reduction in the number of sectors from 496 in the Department of Commerce tables to 466 in IMPLAN.

The matrix is a highly disaggregated representation of national average sectoral input and output technology, and it is on the basis of these production functions that regional purchase patterns are estimated.

Estimates of economic activity and production employment for each of the 466 sectors for all states and every county within each state were made for the components of an input-output table. The estimates of economic activity for states and counties were made through a "downward movement" approach beginning with total national activity, and disaggregating to states and ultimately to counties with control totals employed at each level. As previously noted, the updated 1977 national table was benchmarked with the 1977 National Income and Product Accounts. Since comparable accounts are not available for states or counties, the most suitable regional measures of economic activity were used to disaggregate the national production and demand activity, first among states and then among counties within each state.

Gross output and employment estimates utilized several sources, principally censuses. For example, agriculture sector activity used the Census of Agriculture (U. S. Department of Commerce 1977a) and Agriculture Statistics (U.S. Department of Agriculture 1979).

Gross output measures for most other sectors* (U. S. Department of Commerce 1979a) utilized proxy measures derived from employment and payroll data, principally the national summaries of the County Business Patterns (U. S. Department of Commerce 1977c) and employment data from the Dun and Bradstreet Corporation (Dunn and Bradstreet Corporation 1977).

*Bureau of Economic Analysis input-output sectors 3.00 through 77.05, excluding sectors 11.00, 12.00, 65.01, and 71.01.

Some sectors could be related to specialized data sources such as the Census of Housing (U.S. Department of Commerce 1970) for owner-occupied dwellings, and the Census of Governments (U.S. Department of Commerce 1977d) for government-related sectors. All data was adjusted to the 1977 base year and unreported data was estimated utilizing the RAS procedure.

Final demands were estimated consistent with control totals from the National Income and Product Accounts, by updating the 1963 Multi-Regional Input-Output data (Polenske 1979) using the RAS procedure as suggested by McMenamin and Harring (McMenamin and others 1974). The three components of value added were allocated on the basis of gross outputs. Both final demand and final payment estimates were disaggregated using the "downward movement" approach.

In its entirety, the IMPLAN data base provides a comprehensive, nationwide set of input-output information which can be used to construct nonsurvey-based regional tables. The national technology matrix is maintained at the highly disaggregated 466-sector level of detail, which greatly reduces aggregation errors caused by using 1- or 2-digit SIC (Standard Industrial Classification) industry groupings. Consequently, the industry-commodity relationship is much more consistent than in highly aggregated models.

The hierarchical nature of the data base, achieved by the use of published control totals at each level of disaggregation, results in a data base that permits the construction of models that are consistent both in terms of definition and activity. These principal aspects result in significant improvements over the data used in many previous nonsurvey input-output studies.

Data Reduction: The IMPLAN software system was designed to serve three functions: (1) data retrieval, (2) data reduction and model development, and (3) impact analysis.

The data retrieval system was designed so that the user could have access to input-output data for any U.S. state, county, or combination thereof. The study area data is referenced via a standard set of state and county codes with the extracted data treated as control totals for the region being analyzed. Modification of data, if desired by the user, is permitted.

Utilizing the national technology matrix and the regional control totals, a data reduction method is employed to develop a regional input-output table. The method used exploits the property of "openness" displayed by regional economies compared with the national economy (Richardson 1972).

Regional economies exhibit much greater propensities to import and export than is observed at the national level. Based on the

assumption that trade balances are the principal difference between national and regional purchase patterns (that is, industry production functions are identical, but regional imports and exports make local interindustry transactions different), the supply-demand pool technique (Schaffer and others 1969) for data reduction was adopted.

This method for constructing a regional table begins with the national technology matrix and regional data for gross outputs, final demands, and final payments. Regional data for all 466 sectors is sorted with respect to gross outputs. If the sectoral gross output is greater than zero (firms producing the commodity exist within the region), the corresponding column of direct coefficients is extracted from the national matrix.

Using regional gross outputs and the abbreviated matrix of national direct coefficients, regional purchase transactions are computed. This transactions matrix is then scanned row by row.

If the industry represented by any row has zero regional gross output (indicating that the industry does not occur within the region) the estimated purchases of that commodity are assumed to be non-competitive domestic imports and are shifted from the regional transactions matrix to final payments.

If the gross output is positive and the commodity balance shows a surplus*, the domestic import purchases are assumed to be zero, the regional transactions estimated with national direct coefficients are left unchanged, and the surplus is assumed to be domestic exports.

If the commodity balance indicates a deficit, the regional final demands and transactions estimated with the national coefficients are proportionately reduced across the row to obtain a balance, and the differences assumed to be competitive domestic imports.

The result of this process is a matrix of local transactions between regional industries, plus estimates of both competitive and noncompetitive imports as well as exports.

The data reduction procedure used in IMPLAN produces a complete table of regional input-output accounts.

In addition to this typical table of accounts, detailed reports of sectoral competitive and noncompetitive import purchases are given. Based upon the regional accounts, the predictive input-output model can be derived by computing the standard Leontief-type inverse and calculating various income and employment multipliers.

If appropriate, the number of sectors in the model can be

*Regional gross output is greater than regional final demand plus intermediate demand, estimated with national direct coefficients.

reduced through aggregation prior to inverting the matrix.

Several limitations to nonsurvey data reduction techniques have been noted (Richardson 1972, Fisch and others 1978). The supply-demand pool procedure likewise has limitations. One principal limitation of the supply-demand pool technique is that cross-haul conditions are ignored, while evidence suggests that this may be a common occurrence in regional economies. This arises from the technique's method of allocating local production to meet local requirements before imports or exports are estimated.

Through the use of a highly disaggregated technology matrix and a consistent data base, the IMPLAN system has mitigated though not eliminated many limitations noted by others. For example, Richardson (1972) commented that the use of the national technology matrix may overestimate the interdependence of a regional economy. Similarly, Miernyk (1965) criticizes the supply-demand pool technique assumption of proportionate imports by all purchasing industries. Continued improvements are being sought to enhance the system.

Estimating the regional economic impacts of disturbances in the final demand sector caused by resource management actions is the most frequently used form of input-output analysis employed in Forest Service planning studies. These demand disturbances arise from such activities as changes in timber harvesting, grazing, and recreation, as well as direct budgetary expenditures for goods and services. Economic impacts are expressed by the changes in regional income and earnings, employment, gross output, and various other parameters.

Input-output models are typically used in Forest Service planning studies to estimate the regional economic effects of implementing alternative management plans. These plans describe the intended management activities on a National Forest, along with the expected outputs, resource uses and budgetary expenditures.

Economic impacts are characterized as changes (increases or decreases) from current conditions. Planning teams frequently employ input-out models in other ways. The models provide excellent descriptions of regional economic structure, giving planning teams valuable information for formulating agency policies regarding economic growth or stabilization.

The linkages between Forest Service management actions and corresponding estimates of net changes in regional final demands are critical components in the use of input-output analysis for impact estimation. These disturbances in final demand arise from two principal sources: public expenditure effects and private sector output effects (Cartwright 1979).

Public expenditure effects arise from demand disturbances

caused by government purchases of goods and services. For example, timber stand improvement projects or the construction of recreational facilities involve purchases for labor, materials, and so forth, which can directly be transformed into a demand disturbance vector.

Private sector output effects are somewhat more complex. These effects stem from the use of forest resources and indirectly (from the viewpoint of the Forest Service) result in demand disturbances. For example, the Forest Service's provision of various "factors of production" such as stumpage for wood products, water for municipal and domestic uses, and forage for red meat production must be traced to its final regional economic use, either directly to exports or via "forward linkages" and "stemming-from" effects (see, for example, Roesler and others (1968)).

The effects of the use of forest resources for recreation can be directly transformed into demand disturbances by deriving a typical "bill of goods" purchased locally by the recreationist during the pursuit of such activities. In all cases the demand disturbances represent regional market transactions expressed in purchaser's prices with appropriate transportation and trade margins.

Traditional applications of input-output models, utilizing demand disturbances as the source of interindustry effects, contain an implicit assumption of sufficient resource supply to permit attainment of an equilibrium economy. As is often the case with forest resources, some of the primary resource supplies may be restricted within a regional economy (for example, the amount of water may be restricted).

If the change in forest output is used, under these circumstances, to derive a disturbance in demand and the model used to estimate the resultant multiplier effects, the backward linkages would usually indicate a total demand for the resource exceeding the original change. The IMPLAN system has been designed to perform analyses under these conditions by permitting the user to link the change in resource output directly to change in sectoral gross output rather than a change in final demand.

The input-output model is then used to estimate the maximum level of delivery to regional final demands attainable given the constrained level of gross output. This kind of analysis is often applicable to economies that are highly dependent upon primary resources.

The economic effects estimated with IMPLAN are described by parameters typical of input-output studies. They are structural in nature, permitting multiplier effects to be traced throughout the various regional sectors. Direct, indirect, and induced changes in gross outputs and final demands, employment and import requirements, income and earnings are the most representative parameters used to

describe impacts.

The availability of a complete table also permits calculation of gross regional product. Induced effects are computed using a modified "Type III" multiplier procedure (Miernyk 1965), iteratively solving the open model to capture the effects of induced consumptive spending.

Detailed employment analysis is possible by tracking employment requirements among various occupations, and accounting for the effects of either immigration of workers or reemployment of unemployed local labor. In combination, this information provides a comprehensive, detailed account of potential regional economic impacts.

Use of IMPLAN in Estimating Vegetation Management Effects

Effects on the local economy from timber outputs, permitted livestock grazing, and Forest budgets were developed using IMPLAN. These three "effect groups" were used because they vary significantly among the alternatives and because they were thought to have the potential for significant effects themselves. The IMPLAN model was run separately for Oregon and Washington. Table B-5 shows the response coefficients generated. The process involved estimating the dollar

Table B-5

Relationship of Jobs to National Forest Effects

	<i>Oregon</i>	<i>Washington</i>	<i>Total</i>
Permitted Livestock Grazing (Per 1,000 Animal Unit Months)			
Direct	0.12	0.12	0.12
Indirect	0.32	0.28	0.30
Induced	0.22	0.21	0.22
Total	0.66	0.61	0.64
Forest Budgets (Per Million \$)			
Direct	16.26	12.53	14.40
Indirect	5.63	4.15	2.89
Induced	19.97	17.28	18.63
Total	41.86	33.96	37.91
Timber Offerings (Per Million Board Feet)			
Direct	4.71	4.53	4.62
Indirect	4.42	4.30	4.36
Induced	7.79	8.46	8.13
Total	16.92	17.29	17.11

values that would be injected into the local economies with a change in timber offerings, permitted livestock use, or Forest budget levels.

Direct jobs (and personal income) are jobs in those sectors of the economy that realize the increase in sales. For instance, money is injected into the local economy in the case of timber sale offerings when the timber has been manufactured into its final form and sold. Direct jobs are those in manufacturing firms that make those sales.

Indirect jobs (and personal income) are those associated with firms that supply the wood processing sector such as logging contractors. The term induced refers to the job and income effects generated when those employed directly or indirectly then respend their money in the local economy.

"Jobs" includes full- and part-time positions, temporary and permanent positions without discrimination.

Table B-6

Personal Income Coefficients: Relationship of Personal Income to National Forest Effects (1977 Million \$ – 1987 Million \$)

	<i>Oregon</i>	<i>Washington</i>	<i>Total</i>
Permitted Livestock Grazing (Per 1,000 Animal Unit Months)			
Direct	.0005—.0009	.0005—.0009	.0005—.0009
Indirect	.0033—.0058	.0030—.0053	.0032—.0056
Induced	.0025—.0044	.0025—.0044	.0025—.0044
Total	.0063—.0111	.0060—.0106	.0062—.0109
Forest Budgets (Per Million \$)			
Direct	.2053—.3624	.1574—.2778	.1814—.3202
Indirect	.0813—.1435	.0623—.1100	.0718—.1267
Induced	.2252—.3975	.2046—.3611	.2148—.3791
Total	.5118—.9033	.4243—.7489	.4681—.8262
Timber Offerings (Per Million Board Feet)			
Direct	.0824—.1454	.0847—.1495	.0836—.1476
Indirect	.0661—.1167	.0691—.1220	.0676—.1193
Induced	.0878—.1550	.1002—.1769	.0940—.1659
Total	.2363—.4171	.2540—.4483	.2452—.4328

1977 figures were converted to 1987 figures using the Gross National Product Implicit Price Deflator.

"Personal Income" is not differentiated based on the source of the income.

There is variation in the labor and income components of the different methods of vegetation management. Manual treatment is more labor intensive than is the aerial application of chemicals.

In relation to total area personal income, National Forest-related personal income associated with vegetation management practices accounts for some two percent of the total. Table B-7 summarizes the findings.

Table B-7

Forest-Related Personal Income in Relation to Area Totals
(Includes Only Those Programs Directly Affected by
Vegetation Management)

	<i>Oregon</i>	<i>Washington</i>	<i>Total</i>	<i>National Forests</i>
Million \$	24,634	42,118	66,752	1,208
Percentage of Total	37	63	100	2

Source for Oregon and Washington data is the Statistical Abstract of the United States 1984 published by the U.S. Government Printing Office. Figures shown are for 1980.

National Forest estimates were calculated using a likely future scenario for Forest outputs and the IMPLAN coefficients shown in this Appendix. It is important to remember that these figures exclude Forest employment associated with those outputs and activities with no significant relationship to vegetation management. They do, however, include all personal income associated with National Forest timber offerings, the permitted livestock grazing program, and Forest budgets. Forest figures are in 1977 dollars. Converting them to 1980 dollars would not change the two percent (rounded) figure.

Uncertainty in the Analysis

It is important to "...recognize that all unit values, and especially future values, are only approximations of the worth of the outputs, and are used to assist in placing relative priorities on plan or project alternatives, along with numerous other criteria." (FSM 1971.63—1.d.)

Nearly all data used in this analysis are average (arithmetic mean) values or are derived from averages. This use of averages to represent the National Forests is necessary because it would be impracticable to physically collect precise data on every acre.

Even if it were possible, each acre would be represented as an average situation. The use of averages means that some areas of the National Forests will have their costs or benefits or other characteris-

tics substantially overstated or understated. Thus a decision to manage or not manage vegetation on a certain land type based on averages is not always correct. That is one reason why we are required to do project level analysis. A much-exaggerated example would be the use of Region-wide averages for stumpage values. Such gross averaging would mask the very differences that the analysis is supposed to identify and consider.

To minimize this problem, information was collected from each of the Pacific Northwest Region's 19 National Forests. This allowed the analysts to recognize and maintain the identity of those characteristics which are relevant to the decisionmaking process. The detail of this stratification approaches the limits of practicality for assembling and analyzing data at the Regional level.

Another area of uncertainty is that which arises in making projections into the future. This includes such things as the growth rates of timber and its projected value. By definition, all projections into the future are extrapolations beyond known data points. The farther the projection is extended, the greater the uncertainty.

As far as efficiency considerations, budget concerns, and local economic impacts, our review showed a relatively low degree of sensitivity to the costs of vegetation management. That does not mean that the Forest Service is not concerned about the costs of vegetation management. Rather, it reflects the fact that vegetation management costs are relatively small compared to other costs involved in Forest Service management.

Increases in vegetation management unit costs which appear huge relative to vegetation management costs still leave the total cost of vegetation management quite small relative to the overall level of costs associated with the relevant output.

National Forest historical data is not free from uncertainty. The Management Attainment Reporting Systems (MARS), for instance, is used to record Forest achievements. Live Timber Offerings are included in MARS Code 17.1; Mortality Offerings in 17.2.

Historically, some of the National Forests have reported merchantable endemic tree mortality as Live Timber Offerings. This is understandable because the material can be processed like live sawtimber. Strictly speaking, however, the material should be included in mortality offerings. This causes concern when comparing future empiric yields (which exclude all mortality volume, no matter how recent the mortality or how valuable the timber) with historical Live Timber Offerings, which include merchantable endemic mortality volume. The amount of mortality volume included is not known, nor can it be retrieved from Forest records that were not set up to track this data for future recall.

Estimates of jobs, personal income, and payments to local governments also have a high degree of uncertainty. All three rely on the base data used to generate timber yields, recreation use, and the like, and therefore share all the uncertainty implicit in those estimates. Beyond those concerns, job and personal income estimates are necessarily approximate in that they rely on the IMPLAN model, which uses 1972 average technological coefficients. Payments to local governments are primarily a function of Forest stumpage values—values which can be modified through changes in the appraisal process, the period allowed a contractor for harvesting the timber, legislative changes, etc..

Probably the single area of greatest interest in the analysis is association that exists between long-term sustained yield timber capacity and allowable sale quantity effects in the early decades.

Basically the changes in vegetation management being considered here involve standing timber inventory only to a limited extent. Those stands are established and either are already or will in the future be ready for commercial harvesting, regardless of which alternative is selected for vegetation management. It is those stands that the Forest Service is relying on to make up the overwhelming bulk of its sale program in the early decades.

To some extent, these existing stands could be affected by the threat of wildfire if an alternative were to be selected which so restricted slash treatment as to significantly increase the likelihood of losses to wildfire. In some alternatives that threat is a very real one, but, in general, expected losses are not expected to vary greatly among the alternatives. Any losses in existing timber inventory could reasonably be expected to translate into losses in the allowable sale quantity in the first decades.

Of far greater concern is the question of how future timber yields will be affected on those lands whose reforestation and release has been detrimentally affected. Here though, the relationship between long-term sustained yield timber capacity and the allowable sale quantity in the early decades is not determinate.

It depends on the relationship that exists on each Forest between its established timber inventory and allowable sale quantity schedule. A Forest which expects to see its allowable sale quantity to stairstep upward in future decades as more productive stands replace slow-growing older stands could likely suffer a significant falldown in yields on those future stands without seeing a decrease in allowable sale quantity in the early decades.

For such a Forest, losses in stocking level or slower growth due to the presence of competing vegetation might simply mean that in the distant future, its harvest level might not increase as much as it other-

wise would have.

Other Forests however, are operating very near to the level of their long-term sustained yield timber capacity. That is, their allowable sale quantity is very near to their long-term sustained yield timber capacity. For those Forests, a falldown in future yields would likely translate directly and proportionately into falldowns in the allowable sale quantity even in the early decades.

Another instance is the case of those Forests which are anticipating departing from the policy of nondeclining evenflow. Those Forests may see disproportionately large falldowns in their early decade allowable sale quantities resulting from losses on their future stands.

Adding further to the level of uncertainty is the continuing swirl of social, economic, and political aspirations of the nation, which itself continues to change.

The single feature most responsible for the outcome of the economic analysis is the set of estimates regarding the effects of the alternatives on the allowable sale quantities (ASQ's). To gauge the full extent of that dependence, a sensitivity test was conducted to see exactly how much the model was driven by those estimates.

If disproportionately large changes occur from relatively small changes in an input datum, the model would be said to be highly sensitive to that bit of input. If on the other hand, large changes could be made in that input with little or no change in the model's output, then we would deduce that the model was relatively insensitive to that input. Sensitivity tests thus can be used to highlight areas of great concern.

Table B-8 shows the cause of changes in economic efficiency—allowable sale quantity effects and changes in vegetation management practices.

It shows for instance that PVB is expected to increase in Alternative G by some \$518 million, due to increases in the level of timber production under that alternative. Costs associated with that increase in ASQ are estimated at \$226 million. The overall ASQ effect is therefore clearly beneficial for Alternative G, with almost a 2:1 incremental Benefit:Cost Ratio. Costs tied directly to vegetation management increase markedly in their aggregate (\$275 million) in response to the theme of the alternative.

The table shows rather clearly that the costs and benefits associated with the ASQ clearly dominate the economic analysis.

Sensitivity Analysis

Table B-8

**Economic Criteria Response to Changes in Allowable Sale
Quantity Effects**
(1987 Million Dollars)

	A	B	C	D	E	F	G
<hr/>							
Total Change in PVB from Alt. B¹	-484	REF.	-7,326	-879	-290	-852	+525
Due to ASQ Effects	-484	REF.	-7,314	-878	-290	-845	+518
Due to Veg. Mgt. Practices²	0	REF.	0	0	0	0	0
<hr/>							
Total Change in PVC from Alt. B	-16	REF.	-3,449	-633	-158	-531	+501
Due to ASQ Effects	-98	REF.	-1,942	-288	-100	-301	+226
Due to Veg. Mgt. Practices	+82	REF.	-1,507	-345	-58	-230	+275
<hr/>							
Total Change in PNV from Alt. B	-468	REF.	-3,877	-246	-132	-322	+24
Due to ASQ Effects	-386	REF.	-5,372	-590	-190	-544	+292
Due to Veg. Mgt. Practices	-82	REF.	+1,495	+344	+58	+222	-268

¹ Components may not sum to the Total due to exclusion of livestock category.

² Vegetation management is an activity in support of a benefit.

REF. is the expected situation with implementation of the Forest Plans currently being developed.

Table B-9 shows what would happen if the ASQ effect were only fifty percent of that which we used in our analysis. The analysis answers the question, "What if we overestimated the effect on the ASQ by a factor of two?" The results are striking.

From an efficiency point of view, Alternative C gives up too much timber harvesting. That reduction in timber harvesting overwhelms any reduction in costs.

Alternatives B, E, and A likewise fail to move in the overall rankings of PNV. They still hold positions 2, 3, and 6, respectively. Alternative F moves up slightly in PNV rank from the fifth spot to the fourth. The big changes come with Alternatives G and D. G drops from first place to fifth. D moves from fourth to first.

These shifts occur for a number of reasons. The most obvious is that the alternatives' PNV's are closely ranked—with the exception of C—and the sensitivity test was applied uniformly across the alterna-

tives. Small changes in the rankings are therefore expected.

Alternative G as originally constructed is the only alternative that projects an increase in ASQ from that estimated for Alternative B. This increase in ASQ enabled the alternative to overcome some substantial increases in vegetation management costs associated with the alternative. Without the full extent of the ASQ increase, the increased vegetation management costs overwhelm the anticipated benefits. That accounts for its sharp decline in the sensitivity test.

In the case of Alternative D, it anticipated a sharp net decline in the costs of vegetation management. If this shift in vegetation management practices does not result in the predicted falldown in allowable sale quantity, the alternative looks attractive indeed from an economic efficiency point of view.

Table B-9

Economic Criteria Response to Changes in Allowable Sale Quantity Effects
(1987 Million Dollars)

	A	B	C	D	E	F	G
<hr/>							
Total Change in PVB from Alt. B¹	-242	REF.	-3,669	-440	-145	-429	+266
Due to ASQ Effects	-242	REF.	-3,657	-439	-145	-422	+259
Due to Veg. Mgt. Practices²	0	REF.	0	0	0	0	0
<hr/>							
Total Change in PVC from Alt. B	+66	REF.	-2,478	-489	-108	-380	+388
Due to ASQ Effects	-16	REF.	-971	-244	-50	-150	+113
Due to Veg. Mgt. Practices	+82	REF.	-1,507	-345	-58	-230	+275
<hr/>							
Total Change in PNV from Alt. B	-308	REF.	-1,191	+49	-37	-49	-129
Due to ASQ Effects	-226	REF.	-2,686	-295	-95	-272	+9
Due to Veg. Mgt. Practices	-82	REF.	+1,495	+344	+58	+223	-120

¹ Components may not sum to the Total due to exclusion of livestock category.

² Vegetation management is an activity in support of a benefit.

REF. is the expected situation with implementation of the Forest Plans currently being developed.

Appendix C

Herbicide Use and Efficacy

C

Appendix C

Herbicide Use and Efficacy

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Picloram	C-5
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Hexazinone	C-9
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Issues and Programs

Issues Issues raised during scoping pertain to the amount of use and relative value of herbicides in meeting management program objectives. These include concerns such as:

- the effectiveness of control of important target plants;
- the relative increase or decrease of compound use;
- the primary methods of application and formulations available;
- the management programs with large historical herbicide programs;
- procedures for incorporating new products which display potential effectiveness; and
- hazards to nontarget vegetation and label constraints which limit product value in the forest setting.

The Operational Program The years 1982 and 1983 provide a baseline for an operational program, approximating what may occur under Alternative B. The 16 herbicides addressed in this document account for essentially all of the herbicide use during the 1982 to 1983 seasons.

Table C-1

Typical Annual Acres Treated by Management Program

<i>Application Method</i>	<i>Silviculture</i>	<i>Noxious Weeds</i>	<i>Rights-of-Way</i>	<i>Facility Maint.</i>	<i>Range Mgmt.</i>	<i>Total</i>
<i>Aerial Treatments</i>	16,500	100	300	0	1,750	18,650
<i>Ground Treatments</i>	14,500	1,400	4,200	125	1,750	21,975
<i>Total</i>	31,000	1,500	4,500	125	3,500	40,625

Herbicide Descriptions

Compounds are described in descending order of use in the 1982-1983 program.

Table C-2

Forest Service Herbicide Use Before 1982, Pacific Northwest

	<i>Herbicide</i>	<i>Percent of Treated Acreage</i>
Major use	2,4-D	Approx. 38%
	Glyphosate	Approx. 31%
	Picloram	Approx. 8%
	Triclopyr	Approx. 7%
Minor use: (In order of approximate usage)	Dalapon	Less than 5%
	Atrazine	Less than 5%
	2,4-DP	Less than 5%
	Hexazinone	Less than 5%
	Fosamine	Less than 5%
	Dicamba	Less than 5%
	Asulam	Less than 5%
	Tebuthiuron	Less than 5%
	Diuron	Less than 5%
	Simazine	Less than 5%
	Bromacil	Less than 5%
	Amitrole	Less than 5%

2, 4-D

TRADE NAMES: Weedone, Esteron, DMA-4, Formula 40, Weedar-64, and numerous others.

CHEMICAL NAME: (2,4-dichlorophenoxy) acetic acid.

USE PATTERN: 2,4-D has been heavily used in the silviculture program (Plantation site preparation and conifer release) and has also seen regular use in the right-of-way maintenance and noxious weed control efforts. 2,4-D is often used in combination with other compounds—such as atrazine, dalapon, 2,4-DP, or picloram—to afford a greater span of control. 2,4-D has long been (since the mid-1940's) a favored compound in other agriculture and forestry. A principal advantage is its relative selective control of broad-leaved weeds and woody shrubs, but a nontoxicity to most grasses. It is also less costly than several other herbicides which can be used in a similar vegetative composition.

The use of 2,4-D is decreasing proportionately in recent years as newer materials showing a greater degree of effectiveness in controlling, specific target pests have gained registration for forestry application. 2,4-D, however, continues to be a herbicide of choice in a large number of chemical-use projects.

APPLICATION METHODS AND MODE OF ACTION: 2,4-D is a translocated phenoxy herbicide used in post-emergence applications. Uptake into plants occurs through foliage, and through stems to a lesser degree. It is translocated within the plant to regions of active growth, where it interferes with normal processes of cell wall development.

It is most often applied in a low volatile ester formulation by aerial or ground-backpack methods. Stump or frill treatments of an amine salt formulation can also be effective on larger brush or weed trees.

Typical rates of application have been 1.5-4 lbs. A.E. per acre.

TARGET VEGETATION: 2,4-D has been highly effective in the control of many broadleaved weeds and herbaceous plants. It has also shown consistently good results with red alder, thimbleberry, and serviceberry. With the proper combination of seasonal timing and plant phenology, successful but less consistent results can also be seen with deerbrush, ceanothus, snowberry, manzanita, Pacific madrone, and hazelbrush.

Cut stump or frill injection of 2,4-D amine has resulted in the initial kill and inhibition of sprouting species, such as red alder, California blackoak, madrone, willow, and chinkapin.

POTENTIAL NONTARGET PLANT EFFECTS OR USE LIMITATION: Several conifer species, ponderosa pine in particular, can be sensitive to 2,4-D damage if active tree growth is occurring. In conifer release, the herbicide must be applied when seedlings are dormant and brush is still growing. This period of opportunity will occur only during late spring and early fall. Even with carefully controlled project administration, there may be incidental conifer damage because of different phenological development of individual seedlings in the general population, or changes in aspect or microsite, for example.

Because the mode of entry is primarily through foliage, and then translocation within the plant, it is often advantageous to use mid-range application rates if sprouting inhibition is important. This can help avoid the occasional problem with a too-rapid foliage kill and drying which can limit translocation of the chemical.

Glyphosate

TRADE NAMES: Roundup, Rodeo

CHEMICAL NAME: N-(phosphonomethyl) glycine

USE PATTERN: Glyphosate has been regularly used in the silvicultural program, and has proven effective for right-of-way maintenance, noxious weed control, and facilities maintenance. It is considered a

broad-spectrum, relatively nonselective herbicide. It is one of the newer chemicals, having been first registered for use in 1974. It is a compound heavily used in agricultural and industrial situations, as well as for forestry. Its ability to control herbaceous vegetation, as well as shrubs, is an advantage in some situations.

The use of glyphosate, relative to other compounds, has been increasing in recent years. It has shown a good record of consistency in meeting prescription objectives.

APPLICATION METHODS AND MODE OF ACTION: Glyphosate is absorbed primarily through plant foliage. The specific mode of action is not entirely clear, but it appears to inhibit plant elongation, inhibit synthesis of essential amino acids, and to disrupt the photosynthetic process.

In forest applications, glyphosate (as Roundup), is applied both aerially and from the ground. Typical rates during 1982-1983 were 3 lbs., A.I. per acre (4.5-2 lbs. A.I. for conifer release).

TARGET VEGETATION: Glyphosate effectively controls many sedges, annual and perennial grasses, and broadleaved weeds. It has shown good results with the following woody brush: deerbrush, ceanothus, blackberry, salmonberry, vine maple, red alder, willow, elderberry, bracken fern, and swordfern. It appears to be a good inhibitor of vegetative sprouting. However, the evergreen shrubs and hardwoods are not affected.

POTENTIAL NONTARGET PLANT EFFECTS OR USE LIMITATION: In conifer release applications, timing is more critical for effective brush control with glyphosate in comparison with some other herbicides, such as 2,4-D.

Picloram

TRADE NAMES: Tordon, Amdon

CHEMICAL NAME: 4-amino-3,5,6-trichloropicolinic acid

USE PATTERN: Picloram has seen regular use in noxious weed control, rights-of-way and facilities maintenance, rangeland improvements, and plantation site preparation. Use in release applications is limited due to the sensitivity of many conifers. It is a broad-spectrum chemical used to control a variety of woody plants, and annual or perennial broadleaved weeds. It can be effective in the control of weeds in grassy areas.

The relative use of picloram, in comparison to other herbicides, remains more-or-less constant.

APPLICATION METHODS AND MODE OF ACTION: Picloram is

absorbed both by plant roots and foliage and translocated in the plant. It accumulates at the site of new growth where it is known to function as a plant growth regulator.

It can be applied by ground or aerially in solutions of liquid formulations applied on foliage. A water soluble amine salt is injected into plants or applied to cut surfaces. In this form, it has been used for elimination of weed trees and in precommercial thinning. Picloram pellets or granules are applied at the base of individual target plants where the active ingredient is leached to the rooting zone. It is sometimes applied in combination with 2,4-D for the improved span of control.

Typical application rates during 1982-1983 were .5 to .75 lbs. A.E. per acre, with a high of 1.08 lbs. A.E.

TARGET VEGETATION: Picloram is highly effective in the control of many noxious weeds, such as Canada thistle, leafy spurge, and Russian knapweed. Shrubs which have shown sensitivity include Scotch broom, salmonberry, snowberry, blackberry, and gorse. Injections and cut surface treatments have proven effective in control of many of the common hardwood species. Picloram can also have value where control of conifers is desired, such as utilities or roadside rights-of-ways.

POTENTIAL NONTARGET PLANT EFFECT OR USE LIMITATION: Picloram is persistent in certain soils because of the slow microbial degradation, particularly in cold climates. Breakdown in sunlight, however, is rapid.

Triclopyr

TRADE NAME: Garlon

CHEMICAL NAME: [(3,5,6-trichloro-2-pyridinyl) oxy] acetic acid

USE PATTERN: Triclopyr has been primarily used in the silviculture program, with application also made for rights-of-way and facilities maintenance. Some potential is also seen for rangeland brush control. It is a selective herbicide used for control of a variety of woody plants and broadleaved weeds. Established grasses are not injured at rates needed for weed and brush control. Garlon is a relatively new product in the mix of forestry herbicides.

Relative to other herbicides, the use of triclopyr has been increasing. This is because of its particular effectiveness in the control of root-sprouting species, and the fact that it was a replacement for 2,4,5-T (no longer registered by EPA) in some situations.

APPLICATION METHOD AND MODE OF ACTION: Triclopyr is

Picloram

Triclopyr

Atrazine

absorbed primarily through the foliage and translocates within the plant stem. It accumulates in meristems, where similar to picloram, it interferes with the normal plant growth responses.

Triclopyr is applied both aerially and ground as an ester formulation; and as a cut surface, injection or basal spray as the triethylamine salt formulation.

Average application rates during 1982-1983 were 4 lbs. A.E. per acre (1.5 lbs. A.E. in release applications), and a maximum of 8 lbs. A.E. per acre.

TARGET VEGETATION: Cut surface or basal spray treatments have been effective on tanoak, Pacific madrone, red alder, live oak, bigleaf maple, California black oak, chinkapin, black cottonwood, and willow. Most conifers are also susceptible to cut surface applications. Spray applications can also be effective in the control of brush species such as tanoak, vine maple, blackberry, serviceberry, deerbrush, and snowbrush ceanothus.

An important feature of triclopyr is the ability to control some of the aggressively sprouting hardwood species.

POTENTIAL NONTARGET PLANT EFFECTS OR USE LIMITATION: Treated areas should not be grazed for one year following application.

Atrazine

TRADE NAME: AAtrex

CHEMICAL NAME: 2-chloro-4-ethylamino-6-isopropylamino-s-triazine

USE PATTERN: Atrazine has seen consistent use in many management programs for the selective control of grasses and forbs. Atrazine has been one of the most widely used herbicides in the U.S. since it's introduction in 1958. It is largely ineffective on woody plants.

Atrazine use has been relatively stable in the relative mix of forestry herbicides. It is often combined with other compounds for a broadened span of control. It displays a synergistic effect in combination with dalapon.

APPLICATION METHODS AND MODE OF ACTION: Atrazine is primarily absorbed through plant roots and translocated throughout the plant. It acts primarily as a photosynthetic inhibitor. Atrazine is used as a pre-emergent and early postemergent herbicide, and depends on adequate moisture to move the active ingredient into the rooting zone.

Atrazine has been applied both aerially and by ground methods. Typical application rates during 1982-1983 were 2-3 lbs. AI per

acre, with a high of 4 lbs. A.I. per acre. Certain right-of-way maintenance situations have seen applications of 12.5-25 lbs. A.I. per acre.

TARGET VEGETATION: Atrazine is highly effective in the control of annual grasses. Only moderate control levels have been shown with perennial grasses and herbaceous vegetation. Canada thistle, leafy spurge, and horsetail are among the weeds susceptible to atrazine.

POTENTIAL NONTARGET PLANT EFFECTS OR USE LIMITATION: The effectiveness of atrazine depends on leaching to the plant rooting zone. Lower rates are needed on coarse-textured soils, while higher rates must be applied on fine-textured soil or soils high in organic matter.

Dalapon

TRADE NAME: Dowpon M

CHEMICAL NAME: 2,2-dichloropropionic acid

USE PATTERN: Dalapon has been used regularly in several management programs for the control of grasses. It is primarily used as a selective, postemergence herbicide in forestry. It has wide use for grass control in agriculture and industry. In the Western states, it is commonly used to control established perennial grasses along ditchbanks and noncrop areas.

Use of dalapon is relatively constant in comparison to other herbicides. It is often used in combination with atrazine, or other compounds.

APPLICATION METHOD AND MODE OF ACTION: Dalapon is readily absorbed through both plant roots and foliage, and then translocated throughout the plant tissues. The mode of action is not well understood, but dalapon acts as an inhibitor of both shoot and root growth.

The water soluble powder formulation of dalapon is applied both aerially and with ground equipment. Typical application rates are 3 to 7 lbs. A.I. per acre in site preparation and conifer release. Somewhat higher rates of 10 to 15 lbs. A.I. per acre are used in the range management and rights-of-way maintenance programs. When used for conifer release it should be combined with atrazine to prevent injury to Douglas-fir.

TARGET VEGETATION: Dalapon can effectively control annual and perennial grasses and some sedges.

POTENTIAL NONTARGET PLANT EFFECT OR USE LIMITATION: Dalapon must be applied when the grass is growing and before the

seed heads form. Results will be poor if the grass is not growing well when application is made.

Livestock cannot be grazed on treated areas during the application season.

2,4-DP

TRADE NAMES: Weedone 2,4-DP, Weedone 170, and others.

CHEMICAL NAME: 2-(2,4-dichlorophenoxy) propionic acid.

USE PATTERN: 2,4-DP has been used, generally in combination with 2,4-D, in the silvicultural and rights-of-way maintenance programs. It has also seen occasional use in the range management program. It is one of the systemic phenoxy herbicides, and shows many similarities to the compound 2,4-D. It is more selective than 2,4-D, however. 2,4-DP has been used effectively on many woody shrubs and broadleaved weeds. As with 2,4-D, it is nontoxic to most grasses.

Use pattern with 2,4-DP is also similar to that of 2,4-D, in that it is declining. This is due to the fact that newer, broader-spectrum products are becoming available, and because it is almost always used in a mixture with 2,4-D. 2,4-DP is less harmful to ponderosa pine than is 2,4-D.

APPLICATION METHODS AND MODE OF ACTION: 2,4-DP is a translocated phenoxy herbicide with uptake primarily through the foliage, although basal stem treatments are sometimes used. After translocation, it interferes with the normal plant growth processes.

In forest applications, 2,4-DP is most often applied as a foliar spray in a low-volatile ester formulation. Typical application rates during 1982-1983 were 2-3 lbs. A.E. per acre, with maximums of 5 lbs. A.E. per acre.

TARGET VEGETATION: 2,4-DP can effectively control many broadleaved weeds and herbaceous plants. It has proven highly effective, in combination with 2,4-D, in the control of Pacific madrone.

Significant injury can also result in application to chinkapin greenleaf manzanita, and oceanspray. Basal treatments have shown good control of chinkapin and to a somewhat lesser degree, of bigleaf maple and cherry.

POTENTIAL NONTARGET PLANT EFFECTS OR USE LIMITATION: As with 2,4-D, most conifers can be sensitive to 2,4-DP damage if active tree growth is occurring.

Hexazinone

TRADE NAME: Velpar

CHEMICAL NAME: 3-cyclohexyl-6-(dimethylamino)-1-methyl-1,3,5-triazine-2,4 (1H,3H)-dione.

USE PATTERN: Hexazinone has been used primarily in the site preparation and conifer release programs, and to a more limited extent in rights-of-way maintenance and noxious weed programs. It is a selective pre-emergent and postemergent herbicide in the triazine family. Unlike other triazines, hexazinone controls some shrubs, as well as grasses and forbs. It is one of the newest compounds to be extensively used in forestry applications.

Hexazinone use, in relation to other herbicides, has been increasing. An advantage in some situations is the good residual activity, with nearly complete control of a site possible for several years.

APPLICATION METHODS AND MODE OF ACTION: Hexazinone is absorbed through both the foliage and roots, with translocation primarily upward in the plant. The precise mode of action is unclear, but hexazinone appears to act as a photosynthetic inhibitor. While rapid contact activity may sometimes occur, it often takes a relatively long time period for damage to appear in the target vegetation.

Applications are made both aerially and with ground equipment. Formulations are available as a water soluble powder or granule form. Typical application rates are 1 to 3 lbs. A.E. per acre, although 6 to 12 lbs. A.E. have been used. Application rates vary according to the soil texture and organic matter content as well as the size and density of brush.

TARGET VEGETATION: Hexazinone effectively controls most broadleaved weeds and grasses. Some specific noxious weeds controlled are Canada thistle and starthistle. It can also effectively control small or young woody shrubs, such as manzanita. Hexazinone is very rate-responsive, depending on the type of target vegetation and the duration of control desired. It has application in plantation release programs, but exposure of conifers should be avoided.

POTENTIAL NONTARGET PLANT EFFECT OR USE LIMITATIONS: Rainfall after treatment (1/4 to 1/2 inch) is necessary to activate hexazinone. Hexazinone should only be used in new plantations when the planting stock is at least 2 years old. Newly planted containerized seedlings should not be exposed to hexazinone, because the potting medium will not adsorb the herbicide, leading to injurious levels of uptake by the seedling.

Fosamine

TRADE NAME: Krenite

CHEMICAL NAME: Ammonium ethyl carbamoglyphosphate

USE PATTERN: Fosamine has had periodic use in the silvicultural and rights-of-way maintenance programs, although it is not currently registered for forestry application. It is a carbamate compound used as a postemergence, growth regulator, contact herbicide.

Its use has tended to decline in relation to other herbicides as newer systemic compounds became available for forestry applications.

APPLICATION METHODS AND MODE OF ACTION: Fosamine is absorbed by buds, stems, and plant foliage. It acts by inhibiting or preventing bud development.

It has been applied in liquid formulation by helicopter, broadcast ground application, and foliar on individual plants. Typical right-of-way concentrations have been 6-10 lbs. A.I. per acre, while release projects have been 3 lbs. A.I. Maximum rate during 1982 to 1983 was 12 lbs. A.I.

TARGET VEGETATION: Fosamine has been effective in salmonberry control. It can also provide satisfactory results in red alder, blackberry, and vine maple.

POTENTIAL NONTARGET PLAN EFFECT OR USE LIMITATION: Plants treated in the summer or fall will not show symptoms until the following spring when they leaf out. Only the portion of plant sprayed will show affects.

Dicamba

TRADE NAMES: Banvel, Banex

CHEMICAL NAME: 3,6-dichloro-0-anistic acid (or 2-methyl-3, 6-dichloro-benzoic acid)

USE PATTERN: Dicamba is a relatively nonselective herbicide used against a variety of broadleaved weeds and brush species. It has been used in noxious weed control, rights-of-way maintenance, range rehabilitation, and plantation site preparation. It is phytotoxic to conifers, and can have particular value in some roadside maintenance situations.

Relative to other herbicides, its use has been fairly modest but stable. The dicamba compounds tend to be used when a variety of weeds and shrubs must be controlled on a site.

APPLICATION METHODS AND MODE OF ACTION: Dicamba has been applied aerially and by ground methods. It is often used as a cut surface injection or stump application. A granule formulation has particular application in noxious weed control situations where individual or scattered groups of plants will be treated. Dicamba is ab-

sorbed rapidly by foliage or roots, and is translocated within the plant. It acts as a plant growth regulator; altering shoot and root development. It also interferes with normal flowering and results in destruction of plant cells.

Rates of application during 1982 to 1983 were normally in the range of .5 to 1.5 lbs. A.I. per acre. The maximum during this period was 8 lbs. A.I. per acre.

TARGET VEGETATION: Dicamba is used against a wide variety of broadleaf weed and brush species. It can be effective in noxious weed control for Canada thistle, Russian knapweed, diffuse knapweed, tansy ragwort, and yellow starthistle. Basal treatments have proven effective in the control of red alder and hazel. Vine maple and willow have been controlled, but at reduced injury levels. It is also used in a formulation with 2,4-D to provide a broad span of control for broadleaved annuals and perennials, as well as woody shrubs.

POTENTIAL NONTARGET PLANT EFFECTS OR USE LIMITATIONS: The lower application rates for dicamba are for selective killing of annual broad-leaved plants, and the higher rated for total weed control.

Tebuthiuron

TRADE NAMES: Graslan and Spike

CHEMICAL NAME: N-(5-(1,1-dimethylethyl)-1,3,4-thiadiazol-2-yl)-N,N- dimethylurea

USE PATTERN: Use of tebuthiuron in the Pacific Northwest Region has been primarily in range management for the control of woody vegetation, while grasses are allowed to continue growth. It is normally used in the dry formulations, as granules or pellets. It acts slowly, with plant death often not occurring for 3 years.

Tebuthiuron use has been limited, but the trend is for an increase relative to other herbicides.

APPLICATION METHODS AND MODE OF ACTION: Tebuthiuron is absorbed through the roots. The mode of action in plants is to inhibit photosynthesis, resulting in repeated defoliation. This eventually depletes carbohydrate reserves and results in the death of susceptible plants.

Applications have been by ground methods, or (rarely) by air (pellets). Average application rates have been .7 to 1.6 lbs. A.I. per acre, with a maximum of 8 lbs. A.I. per acre.

TARGET VEGETATION: Many brush, trees, and perennial broadleaf plants are controlled by tebuthiuron. It can also be used for control of

Dicamba

Tebuthiuron

Asulam

Diuron

annual weeds, some perennial grasses and weeds. Some noxious weeds, such as puncturevine, morning glory, spurge, and Russian thistle are also controlled by tebuthiuron.

POTENTIAL NONTARGET PLANT EFFECTS OR USE LIMITATION:

Tebuthiuron should not be used around desired plants, or where their roots may grow into contact with the chemical. Vertical leaching in the soil is slow and no lateral chemical movement has been observed.

Asulam

TRADE NAME: Asulox

CHEMICAL NAME: methyl sulfanilylcarbamate

USE PATTERN: Asulam is a carbamate compound used as a selective, postemergence, systemic herbicide. It has been used periodically in the silvicultural and rights-of-way maintenance programs for the control of bracken fern, broadleaved weeds, and perennial grasses. It has had occasional use for fencerow and administrative site maintenance.

Asulam has been a minor but relatively stable component of forestry herbicide use.

APPLICATION METHODS AND MODE OF ACTION: Asulam can be applied in a liquid concentrate formulation by either aerial or ground methods. Asulam is absorbed rapidly by plant foliage and roots, and then translocated within the plant. Signs of herbicidal action are yellowing of leaves, stunting of the plant, and death of the actively growing plant points. The visual signs of herbicide activity may not appear for 2 or 3 weeks.

Application rates averaged 3.34 lbs. A.I. per acre, with a high of 7 lbs. A.I. per acre during 1982 to 1983.

TARGET VEGETATION: Asulam can be particularly effective on bracken fern.

Diuron

TRADE NAME: Diuron, DMU, Karmex, and Krovar

CHEMICAL NAME: 3-(3,4-dichlorophenyl)-1,1-dimethylurea

USE PATTERN: Diuron is a substituted urea compound used as a pre- and postemergence herbicide. It is used as a soil sterilant in non-crop areas. It has had periodic use in the rights-of-way maintenance program for total vegetation control. It has fairly widespread use in agriculture for weed control in fruit, nut, and grain crops.

Diuron has been a small but stable component of the rights-of-way maintenance herbicide use program.

APPLICATION METHODS AND MODE OF ACTION: Diuron is rapidly absorbed through the plant root system and translocated to the aerial plant parts. Toxicity is due to the inhibition of photosynthesis, with leaf chlorosis occurring within several days to several weeks, depending on the rate of application used.

Average application rates during 1982 to 1983 were 5 to 10 lbs. A.I. per acre. Label-approved rates of up to 20 to 60 lbs. A.I. per acre are almost never used in forestry situations.

TARGET VEGETATION: Diuron can provide effective control of grasses and broadleaved weeds. Some specific weeds are foxtail, ragweed, ryegrass, and Johnsongrass. It is also used occasionally for control of aquatic weeds in fish hatcheries.

POTENTIAL NONTARGET PLANT EFFECTS AND USE LIMITATION: Diuron is relatively persistent in soils, and under typical soil texture and moisture conditions will not leach more than 6 inches below the soil surface. Microbial decomposition is the main mechanism of decomposition. A susceptible plant species should not be planted or seeded for at least 12 months after diuron application.

Simazine

TRADE NAME: Princep, Aquazine

CHEMICAL NAME: 2-chloro-4,6-bis (ethylamino)-5-triazine

USE PATTERN: Simazine is a selective triazine compound used as a pre-emergent herbicide for the control of grasses (primarily annuals) and broadleaved weeds. It has had minor use in the rights-of-way maintenance and plantation maintenance programs. It has only rarely been used for control of algae and aquatic plants.

Simazine has been a minor component of the herbicide use program. Other grass-killers are normally favored because of the tendency for simazine to bond tightly in the top 2 inches of soil.

APPLICATION METHODS AND MODE OF ACTION: Simazine is absorbed through plant roots, and shows no contact activity. It is translocated in the plant xylem system, where it interferes with a number of plant biochemical processes.

It has been applied by ground spot or broadcast methods. Rates during 1982 to 1983 were at 4 to 4.6 lbs. A.I. per acre.

TARGET VEGETATION: Simazine is effective in the control of annual grasses, and to a lesser extent with perennial grasses and forbs. It is sometimes applied in a mixture with atrazine. It is available in wettable powder, granule, and liquid suspension formulations. Some specific weeds controlled are knapweed, leafy spurge, Canada thistle, and dandelion.

Diuron

Simazine

Amitrole

*Bromacil***POTENTIAL NONTARGET PLANT EFFECTS OR USE LIMITATION:**

Adequate moisture is required to activate simazine. It is relatively persistent, particularly in cold, dry, or infertile soils. It does not prevent germination, but destroys the plant after rooting.

Amitrole

TRADE NAMES: Amitrol-T, Amizol, and Weedazol

CHEMICAL NAME: 3-Amino-1,2,4-triazole

USE PATTERN: Amitrole is a nonselective, post-emergence herbicide which has occasional use in the rights-of-way maintenance program. An advantage of amitrole over some other compounds which afford similar vegetation control lies in product cost. Amitrole was patented in 1954 as a herbicide and plant growth regulator.

Amitrole has been a relatively insignificant compound if the herbicide use program. It is also formulated with simazine for use as a soil sterilant around buildings, storage areas, and parking lots.

APPLICATION METHODS AND MODE OF ACTION: Amitrole is absorbed by leaves and roots, and then translocated by both xylem and phloem systems within the plant. It affects a number of biochemical processes and tends to accumulate at the site of new growth.

Amitrole is generally applied by ground methods, although aerial helicopter application may rarely be used. Rate of application during 1982 to 1983 averaged 2 to 4 lbs. A.I. per acre. The approved label maximum is 20 lbs. A.I. per acre.

TARGET VEGETATION: Amitrole is relatively nonselective. It is particularly effective on annual and perennial grasses, and herbaceous vegetation. Some specific target plants have been poison oak, blackberry, thistles, and leafy spurge. It is effective on many root-suckering species of brush.

POTENTIAL NONTARGET PLANT EFFECTS OR USE LIMITATION: Conifers will be damaged by amitrole. Directions are to keep livestock out of treated areas during the season of use.

Bromacil

TRADE NAME: Hyvar, Urox B

CHEMICAL NAME: 5-bromo-6-methyl-3-(1-methylpropyl) uracil

USE PATTERN: Bromacil has had some limited use in the rights-of-way maintenance program. It is used as a selective herbicide at normal use rates for control of a broad spectrum of broadleaved weeds, grasses, and some woody shrubs.

Bromacil has been a relatively insignificant component of the herbicide use program.

APPLICATION METHODS AND MODE OF ACTION: Bromacil is absorbed by plant roots. Adequate moisture must be available to activate bromacil. Toxicity is apparently related to the inhibition of photosynthesis within chloroplasts.

Applications of the wettable powder formulation have been by ground methods. Right-of-way use has been at the rates of 2 to 8 lbs. A.I. per acre, although a maximum could be as high as 30 lbs. A.I. per acre.

TARGET VEGETATION: Many annual and perennial weeds, and brush species can be controlled with bromacil. Some specific plants include cheatgrass, bracken fern, and foxtail. Some woody shrubs, such as willow and maple, can be controlled by basal spot applications. While absorption is primarily through roots, there is a limited amount of contact activity. This can be greatly increased with the use of a surfactant. It is also available in a formulation with diuron.

POTENTIAL NONTARGET PLANT EFFECTS OR USE LIMITATION: A more prompt plant kill occurs if bromacil is applied during the early part of the growing season. Deep rooted perennials can be difficult to control with diuron. Diuron is relatively slow acting, but seasonal or longer control of undesirable vegetation can be expected.

New Products

Herbicides not included for analysis in this EIS will periodically become available for use in forestry. These may be newly developed products, or the labelling of existing compounds for expanded uses. In this case, the potential use of these tools will be subject to certain process and analysis requirements:

1. NEPA Documentation. An analysis of potential environmental effects, which will be documented in a supplement or addendum to this document.
2. Human Health Risk Assessment—Worst Case Analysis. This is in response to the 1984 U.S. Court for the District of Oregon decision.
3. Decision Notice Signed by the Responsible Official. A decision will be made as to the appropriateness of use, and direction regarding use restraints or mitigation measures to be utilized in project implementation.

There appears to be a general reluctance on the part of chemical manufacturers to develop and register new products for use in forestry. This

is apparently due to the fact that forestry use is a small component of the total pesticide market; and because it is difficult to recover the large front-end investment needed for product development, testing, and registration through the Environmental Protection Agency.

Two examples of herbicides that could be considered for future use on National Forest lands are:

Metsulfuron Methyl (trade name, Escort). This compound has been labelled for use in Washington and Oregon. It is registered for roadside use only, although there is some potential that site preparation use will be approved.

Sulfometuron methyl (trade name Oust). This compound is labelled for use in roadside or utility rights-of-way, and other uses. The primary advantage of these two chemicals is the fact that extremely low doses of active ingredients are needed for vegetation control.

1. The first step is to identify the problem.
2. The second step is to define the problem.
3. The third step is to analyze the problem.

4. The fourth step is to develop a solution.
5. The fifth step is to implement the solution.

6. The sixth step is to evaluate the solution.
7. The seventh step is to monitor the solution.

8. The eighth step is to report the results.
9. The ninth step is to conclude the project.

10. The tenth step is to reflect on the process.

11. The eleventh step is to document the process.

12. The twelfth step is to share the results.

13. The thirteenth step is to celebrate the success.

14. The fourteenth step is to learn from the experience.

Appendix D

Human Health Risk Assessment (Quantitative)

D

Appendix D

to the

Environmental Impact Statements of Management of Competing Vegetation

**Human Health Risk Assessment for the
Use of Herbicides in the
Vegetation Management Programs of the
U.S. Forest Service in
Washington and Oregon and the
Bureau of Land Management in Western Oregon**

**Prepared for the USDA Forest Service
and the USDI Bureau of Land Management**

by

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Appendix D
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Section 1

Section 1

INTRODUCTION

PURPOSE

The purpose of this analysis is to assess the risk to human health of using 16 different herbicides for vegetation management on Forest Service lands located in Washington and Oregon and on Bureau of Land Management (BLM) lands in western Oregon. This risk assessment is a supplement to the Forest Service Environmental Impact Statement (EIS) entitled Methods of Managing Competing Vegetation: A Programmatic Environmental Impact Statement (1981) and the BLM EIS entitled Western Oregon Program: Management of Competing Vegetation (1983). Both EIS's analyzed the environmental impacts of using various alternatives for managing competing vegetation in the Pacific Northwest.

OVERVIEW OF THE RISK ASSESSMENT

This risk assessment examines the potential health effects on all persons who might be exposed to any of the 16 herbicides as a result of activities related to the vegetation management programs. People potentially at risk are separated into two categories. The first group--workers--includes applicators, supervisors, and other personnel directly involved in the application of herbicides. The second group--the public--includes forest visitors or nearby residents who could be exposed through the drift of herbicide spray droplets, through contact with sprayed vegetation, or by eating vegetation (such as berries), game, or fish containing herbicide residues or drinking water that contains such residues.

The analysis of the potential human health effects of the use of chemical herbicides for BLM's management of vegetation in western Oregon was accomplished using the methodology of risk assessment generally accepted by the scientific community. In essence, pesticide risk assessment consists of comparing doses people may get from applying the pesticides (worker doses) or from being near an application site (public doses) with doses shown to be safe in animal laboratory studies.

A number of factors contribute to the uncertainty in this process of judging risks to human health from laboratory animal studies. First, the safe levels established in the laboratory are the result of tests on laboratory animals, particularly rats and mice, where dose levels produce no observed effects. To allow for the uncertainty in extrapolating from these no-observed-effect levels (NOEL's) in lab animals to safe levels for humans, additional safety factors are used. The generally accepted factors (NRC 1986) are 10 for moving from animals to humans (between species

variation) and another 10 to account for possible variation in human responses (within species variation). This 10 times 10 or 100-fold safety factor means the laboratory NOEL dose reduced one hundred fold would normally be considered a safe dose. In this risk assessment a margin-of-safety (MOS) has been calculated for each estimated dose by dividing the animal NOEL by the estimated dose. The computed MOS is then compared to the 100-fold safety factor to judge the risks of toxic effects.

A second area of uncertainty is in judging the risk to humans of doses that may be received once or perhaps a few times in a person's life (accidental worker doses and all doses to the public fall in this category) by comparing those human doses to levels of the chemical that produced no ill effects in laboratory animals even though the animals received the doses every day of their lives. This risk assessment uses the MOS approach discussed above in comparing one-time human doses to lifetime animal doses in all of these cases even though this leads to an exaggeration of the risks.

A different approach is used to assess the risks to humans of chemicals that may cause cancer since they are assumed to have no comparable margin of safety so that there is some risk even at extremely low doses. In this case a cancer potency value, expressing the probability of developing tumors at increasing dose levels, is taken from lab animal studies and adjusted for the differences in body weight and lifetime duration between the lab animals and humans. This potency times an estimated human lifetime dose provides an estimate of human cancer risk.

A third area of uncertainty involves the estimation of the human doses liable to occur in herbicide use. This risk assessment has been designed to overestimate doses to err on the side of safety. In reality, workers are likely to receive some low level doses because they work with the chemicals routinely. However, standard safety practices and the use of protective clothing will normally reduce their actual dose levels far below those estimated in this analysis. The same is true of the doses from any spraying or spill accidents that might occur, since the normal procedure would be to wash immediately. In addition, no member of the public is likely to receive as high a dose as estimated in this risk assessment; again because normal safety practice and the remoteness of most treated areas limit the possibility of the public receiving any dose at all. Furthermore, the public doses estimated here exaggerate the amount they could receive. No herbicide degradation is assumed to occur, the public is not assumed to wash themselves or their food items after a spraying, and they are assumed to consume water that has received herbicide from drift or a spill immediately after the event. Thus, the way in which exposures are estimated in this risk assessment and the way the risks are judged both tend to exaggerate the real risks.

The risk assessment includes analyses of a range of possible exposures--from realistic to worst case--resulting from herbicide application. Typical application scenarios (routine-realistic) are used to estimate the doses to workers and to members of the public who may be nearby that may reasonably be expected to occur during routine operations. Extreme application scenarios (routine-worst case) are used to give very high dose estimates that are

not likely to be exceeded except in the case of an accident. Accident scenarios (accidental-worst case) are used to estimate doses to workers and the public that may result from direct exposure to the herbicide spray mix or concentrate or from drinking water into which a truckload of herbicide mixture or a drum of herbicide concentrate has been spilled.

Structure of the Risk Assessment

This risk assessment employs the three principal analytical elements described by the National Research Council (1983) as necessary to characterize the potential adverse health effects of human exposures to existing or introduced hazards in the environment: hazard analysis, exposure analysis, and risk analysis.

1. **Hazard Analysis** requires gathering information that is used to determine the toxic properties of each herbicide. Human hazard levels are derived primarily from the results of laboratory experiments on animal models, such as rats, mice, and rabbits, supplemented where appropriate with information on human poisoning incidents, field studies of other organisms, and data on chemical structure.
2. **Exposure Analysis** involves estimating single and multiple exposures to persons potentially exposed to the herbicides, determining the doses likely to result from those estimated exposures, and determining the number and characteristics of persons in the exposed populations.
3. **Risk Analysis** requires comparing the hazard information with the dose estimates and the probability that they could occur to predict the health effects to individuals under the given conditions of exposure.

The relationships among these three components are illustrated in Figure 1-1. This risk assessment identifies uncertainties, such as areas where scientific studies are unavailable, and presents the results of all worst case analyses. The discussion that follows describes briefly how each component in the structure was addressed in this risk assessment.

Hazard Analysis

The 16 herbicides being considered by the Forest Service and BLM in their vegetation management programs are amitrole, asulam, atrazine, bromacil, 2,4-D, 2,4-DP (dichlorprop), dalapon, dicamba, diuron, fosamine, glyphosate, hexazinone, picloram, simazine, tebuthiuron, and triclopyr. The hazard involved in the use of each of the herbicides was determined in a thorough review of available toxicological studies. Where no studies have been conducted in a particular area, for example, mutagenicity, these areas are identified and a worst case analysis is conducted in Section 5. Scientific uncertainty regarding the results of these studies, for example, concerning the results of the cancer studies on glyphosate and 2,4-D, is also discussed. The hazard analysis is discussed in Section 3.

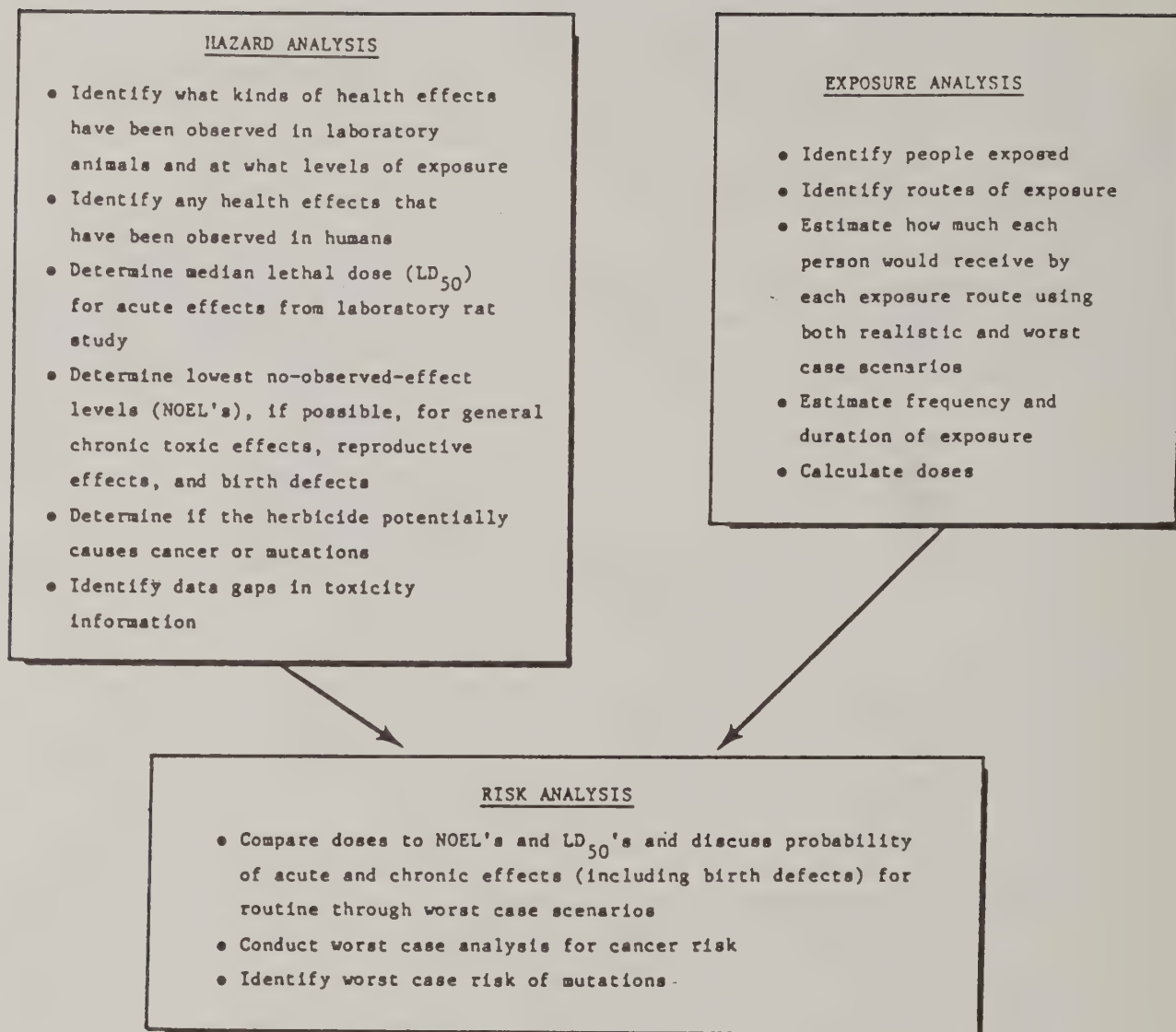


Figure 1-1 Components of the Risk Assessment Process

The toxicological data base for each herbicide was reviewed for acute and chronic effects on test animals. Toxicity information is summarized for 12 of the 16 herbicides in the background statements of Forest Service Agricultural Handbook No. 633 (USDA, 1984). Tebuthiuron toxicity is reviewed in a background statement prepared for the Forest Service as a supplement to Handbook No. 633. Toxicity information is summarized for the herbicides asulam, diuron, and bromacil in background statements written in conjunction with this risk assessment. These documents are incorporated by reference into this Supplement in accordance with 40 CFR 1502.21 and are available for review at all Forest Service and BLM District Offices in Oregon and Washington as well as at the address shown on the cover page.

Exposure Analysis

To estimate the potential human exposures to the 16 herbicides, the various aspects of the vegetation management programs of the Forest Service and Bureau of Land Management in Washington and Oregon that employ herbicides to control vegetation were examined. The major aspects of the vegetation management programs that determine the potential levels of herbicide exposure were identified, including human activities associated with or in proximity to treatment areas, application methods, application rate, size and configuration of spray areas, project design features, and mitigation measures.

Herbicide Spraying Operations

The 16 herbicides examined in this risk assessment are applied aerially, using fixed-wing or helicopter aircraft, or on the ground, using trucks or tractors, backpack sprayers, or handheld application devices. Table 1-1 shows the types of operations where the herbicides are used and the approximate yearly acres affected in Region 6 for the Forest Service and in western Oregon for BLM. The cumulative analysis makes the worst case assumption that 100,000 acres are treated each year as a result of the combined programs. The size of the program and the mix of activities may vary in any given year as described in each parent EIS.

These annual programs would involve a limited number of large projects and many small projects ranging from one to many separate treatment units. Individual silviculture treatment units within a project typically range from 15 to 60 acres. A number of individual sites are normally treated at one time, with 120 to 150 acres normally treated per day. Occasionally there are treatment areas much smaller (less than 1 acre) or much larger (up to 200 acres), especially on wildlife rehabilitation projects. Treatment units for range management projects are generally larger, with 200 to 400 acres normally treated each day.

More than 100 projects, with treatment units ranging in size from less than 1 acre (for facility maintenance) to 400 acres for range management, occur annually on BLM lands in western Oregon and Forest Service lands within Region 6. The area treated with various herbicides in 1982 was less than 1 percent of the possible 21,746,000 acres of National Forest land in Region 6. Slightly more than 1 percent of the 2,383,000 acres of land administered by BLM in western Oregon was treated with herbicides in 1982. Further details about these operations are given in the parent EIS's and in Section 2.

Affected Populations

In calculating the potential doses to persons at risk from herbicide applications, two populations were considered: workers and the general public. The workers included the personnel directly involved in the spray operations: the mixers and loaders, the truck-sprayer applicators and drivers, the backpack sprayers, the hand applicators, the pilots, the observers, and the supervisors. The public included forest visitors and nearby residents who may be directly exposed to herbicide as a result of

Table 1-1

Herbicide Spraying Operations for Forest Service and BLM Lands

Treatment Operations	Forest Service Acres	BLM Acres
Silviculture: Site Preparation and Conifer Release	27,000 - 35,000	38,800 - 44,100
Right-of-Way Management	3,000 - 6,000	1,900
Noxious Weed Control	1,400 - 1,600	300 ^a
Range Improvement	2,000 - 5,000	---- ^b
Facilities and Recreation Site Maintenance	100 - 150	---- ^b

^aBLM has prepared a separate EIS on noxious weed control. U.S. Department of the Interior, BLM. Northwest Area Noxious Weed Control Program, Draft EIS May 1985.

^bIncluded in BLM's noxious weed control program.

drift, by contact with vegetation that has received herbicide drift, or by being accidentally sprayed. The public may be indirectly exposed by eating food items or drinking water containing herbicide residues.

Routine Exposure Scenarios

This risk assessment examines the health effects of exposure to an individual herbicide treatment as well as the cumulative effects of exposure over a number of years. To represent the range of doses under normal operating procedures, eight application scenarios were used. Under the routine-realistic scenario, four application methods, employing normal herbicide application rates and typical treatment unit sizes, were used to calculate realistic doses to workers. Doses to members of the public who may be in the area or who may live nearby were calculated for aerial, truck, and backpack scenarios. No public exposures were expected from hand-application treatments because drift is negligible from these methods.

Four additional scenarios, using the same application methods as routine-realistic but employing the highest application rates likely to be used and the largest treatment unit sizes under conditions conducive to offsite herbicide drift, were used to estimate routine-worst case doses to workers and

the public. These dose estimates purposely overestimate doses expected from routine applications.

Cumulative doses were estimated by using information on average and maximum treatment days per year and on average and maximum number of years exposed for workers and for the public.

Accident Exposure Scenarios

Because all human activities involve the possibility of error, the use of herbicides in vegetation management involves the possibility that humans may inadvertently receive unusually high exposures to the herbicides because of accidents.

To examine what potential health effects could occur in an accidental situation, a number of accidental-worst case scenarios are analyzed. Exposures analyzed include direct aerial application of herbicide on a person, spills of concentrate or herbicide mix on workers in mixing and loading, and spills of herbicide into drinking water supplies. One accidental scenario assumes that a person enters a treated area (ignoring warning signs) before any herbicide has dried or degraded.

The probabilities of these accidental scenarios range from unlikely to extremely unlikely. Wherever possible, historical records of accidents were used in determining the probabilities of accident occurrence.

Dose Estimation

Estimates of routine doses to workers were derived from field studies on the five herbicides (2,4-D, 2-4-DP, dicamba, amitrole, and picloram) for which that information is available (see Table 4-3). Where no studies existed on a particular herbicide, doses were extrapolated from a 2,4-D worker exposure study that used the same application method.

Worker exposures to each herbicide were based on the worker's task, for example, backpack sprayer, pilot, mixer-loader, and so forth, rather than the type of vegetation management project because the same equipment and procedures are often used in these operations. The exposures between operation types are weighted by application rate and number of hours worked per day. Where the exposure of a worker in a particular task, such as mixer-loader, is significantly different from one project type to another, that exposure is determined separately for each representative operation.

Exposures and doses to members of the general public were derived by using data on herbicide drift from field studies and by applying various assumptions about dermal penetration, amount of skin exposed, and diet. Details of the exposure analysis are given in Section 4.

Risk Analysis

Human health risks of the vegetation management program were evaluated by comparing the doses of workers and the general public calculated for routine operational and accidental exposure scenarios to the laboratory-determined toxicity levels described in the hazard analysis.

Risk of acute and chronic threshold effects is evaluated by comparing estimated doses to toxicity reference levels derived from LD₅₀'s and NOEL's (no-observed-effect levels) in laboratory animal studies, and a margin of safety (MOS) is derived. Risk increases as the estimated dose approaches the laboratory toxicity level.

Nonthreshold risk, that is, the potential for these herbicides to cause cancer and mutations, was evaluated differently. The analysis showed that currently there is scientific uncertainty regarding the potential of five of the herbicides--amitrole, atrazine, picloram, 2,4-D, 2,4-DP, and glyphosate--to cause cancer in humans. Therefore, this risk analysis uses the worst case assumption that these five herbicides would cause cancer in exposed persons. The risk of cancer at a given level of exposure, based on the estimated average daily exposure over a 70-year lifetime, was derived for the herbicide in question from laboratory animal data on tumor incidence at increasing dose levels. These data were corrected for species differences, body size difference, dose frequency, and duration of exposure; and the risk of cancer was calculated for various categories of people that may be exposed to the herbicides.

The risks of heritable mutations are discussed based on available test data on bacteria, yeasts, plants, mammalian cells in culture, and whole animals. Where no test data are available, a worst case assumption is made that these herbicides are mutagenic and that risk is compared to the herbicide's cancer risk.

Cumulative risk for individuals is discussed in terms of lifetime exposures to a given herbicide for workers and for members of the public. Risk of synergistic effects is discussed in terms of the available evidence of enhanced toxicity in mixtures of two or more herbicides. Risk to sensitive individuals is discussed qualitatively in terms of the likelihood of a sensitive individual being exposed.

WORST CASE ANALYSIS REQUIREMENTS

As indicated earlier, this document is a supplement to the Forest Service and BLM Environmental Impact Statements named on page 1-1 and has been prepared pursuant to the requirements of the National Environmental Policy Act (NEPA) and the Council on Environmental Quality (CEQ) regulations for implementing NEPA. The CEQ regulations (40 CFR 1502.22) state:

When an agency is evaluating significant adverse effects on the human environment in an environmental impact statement and there are gaps in relevant information or scientific uncertainty, the agency shall always make clear that such information is lacking or that uncertainty exists.

(a) If the information relevant to adverse impacts is essential to a reasoned choice among alternatives and is not known and the overall costs of obtaining it are not exorbitant, the agency shall include the information in the environmental impact statement.

(b) If (1) the information relevant to adverse impacts is essential to a reasoned choice among alternatives and is not known and the overall costs of obtaining it are exorbitant or (2) the information relevant to adverse impacts is important to the decision and the means to obtain it are not known (e.g., the means of obtaining it are beyond the state of the art) the agency shall weigh the need for the action against the risk and severity of possible adverse impacts were the action to proceed in the face of uncertainty. If the agency proceeds, it shall include a worst case analysis and an indication of the probability or improbability of its occurrence.

This risk assessment identifies a number of uncertainties or data gaps, including the following:

1. Field studies on exposure to workers for all of the herbicides except 2,4-D, 2,4-DP, dicamba, amitrole, and picloram.
2. Information on exposure of the public to the 16 herbicides.
3. Field data on residue levels in plants and animals most likely to be found in and around treatment areas for some of the herbicides.
4. Mutagenicity studies for bromacil, dalapon, and diuron.
5. The potential for amitrole, atrazine, asulam, bromacil, 2,4-D, 2,4-DP, picloram, and glyphosate to cause cancer.
6. Toxicity information on the synergistic effects from exposure to more than one herbicide.

These areas of uncertainty are important in deciding what is the best alternative for action; however, the cost of obtaining this information is an important consideration. From discussions with the Environmental Protection Agency, the Department of Agriculture, the Department of the Interior, and the chemical manufacturers, it is estimated that the costs per chemical of conducting some of the standard laboratory toxicity tests would be \$1.5 to \$2.0 million for a chronic toxicity study with rats and dogs; \$1.5 to \$2.5 million for an oncogenicity test with rats and mice; and \$50,000 to \$100,000 for each mutagenicity and chromosomal study.

(insert Table 1-2)

Table 1-2. Data Gaps Identified in Risk Assessment for Pesticides

DATA GAPS		PESTICIDES*											
		Amitrole ^{1,2}	Atrazine ³	Bromacil ^{4,5}	Dicamba ^{6,7}	Diuron ^{8,9}	Hexazinone ¹⁰	Picloram ¹¹	Simazine ¹²	Asulam ^{13,14}	Glyphosate ¹⁵	2,4-DP ¹⁶	
Product Chemistry													
Product Identify		X	X	X		X	X	X	X				
Analysis and Certification of Product Ingredients		X	X	X	X	X	X	X	X				
Physical and Chemical Characteristics		X	X	X		X	X		X				
Environmental Fate													
Degradation		X	X	X	X	X		X	X				
Metabolism		X	X	X	X	X	X	X	X				
Mobility		X	X	X	X	X	X	X	X				
Dissipation		X	X	X	X	X		X	X				
Accumulation		X	X	X	X	X		X	X				
Residue Chemistry													
Nature of Residue								X	X				
Residue Analytical Method			X			X		X	X				
Storage Stability					X	X		X	X				
Magnitude of Residue			X		X	X		X	X				
Toxicity													
Acute		X				X	X	X	X	X	X		
Subchronic		X			X	X		X	X	X	X		
Chronic		X	X	X	X	X		X	X	X	X		
Oncogenic				X	X	X		X	X	X	X		
Teratogenic		X	X	X	X	X		X	X				
Mutagenic			X		X	X		X	X				
General Metabolism			X			X	X	X	X	X	X		
Ecological Effects													
Avian		X		X	X			X	X				
Aquatic		X		X	X	X		X	X				
Mammalian													
Nontarget Insects													
Spray Drift***								X					
Plant Protection***													
Nontarget area													
phytotoxicity													

*Information not available to determine data gaps for Dalapon, Fosamine, Tebuthiuron, and Triclopyr.

**Information not available to determine data gaps other than toxicological for Asulam, and 2,4-DP.

***These issues were addressed for Picloram only.

Sources: 1EPA, 1983c
 2EPA, 1984t
 3EPA, 1983d
 4EPA, 1985f
 5EPA, 1982d
 6EPA, 1983g
 7EPA, 1983e
 8EPA, 1983h
 9EPA, 1983f
 10EPA, 1982e
 11EPA, 1985g
 12EPA, 1982c
 13EPA, 1983
 14EPA, 1984u
 15EPA, 1985h
 16EPA, 1982b

The estimated costs to fill the specific data gaps listed above are:

1. Worker exposure studies would cost approximately \$200,000 per chemical.
2. No acceptable protocol is available for measuring all of the various routes of exposure of the public, but these studies would be more expensive than the worker exposure studies.
3. The cost of measuring residues in plants and animals would be between \$50,000 and \$100,000 per chemical per plant or animal.
4. The mutagenicity and chromosomal studies for bromacil, dalapon, and diuron would cost approximately \$450,000.
5. The five oncogenicity studies for amitrole, asulam, bromacil, 2,4-D, 2,4-DP, picloram, and glyphosate would cost approximately \$11.2 million.
6. Synergistic studies would be extremely expensive because of the great number of tests that would be necessary; there are 120 combinations of the 16 herbicides if studied two at a time.

The overall cost of conducting the studies to fill the data gaps is considered exorbitant with respect to the limited funds available to the Forest Service and BLM. In addition, the time needed to perform and evaluate most of these tests is more than 2 years and would seriously delay the implementation of the vegetation management programs. Many of the outstanding toxicological studies have already been requested by EPA, and the results of these studies will be considered when they become available. In addition, both agencies have ongoing research and monitoring programs to examine the various aspects of herbicide treatment, and these results will be considered as they become available.

Because the cost of filling the data gaps is considered exorbitant, a worst case analysis was conducted for those areas where information is not available or when there is uncertainty. The probability of a worst case occurring was also estimated. The worst case analyses for the first three uncertainties listed on page 1-8 involved estimating the upper limit of herbicide exposure from routine application procedures and from accidents. The worst case scenarios involving routine herbicide application operations consist of those combinations of parameters, such as treatment unit size, duration of exposure, application rate, application equipment, and meteorological conditions, that give the highest reasonable exposure value. Worst case accidents include direct spills of concentrate on workers' skin, the direct spraying of an individual, and public exposure through drinking water contaminated by a spill.

The worst case analysis for the mutagenicity of chemicals for which there is no data or where there is some positive testing for mutagenicity assumed that all these herbicides could cause mutations. The probability of mutagenic activity was based on available cancer data. The worst case analysis for herbicides that had either positive cancer studies or for which there

is scientific uncertainty assumed that these chemicals could cause cancer. The most conservative cancer potency value for a chemical was computed by using the highest rates of tumor formation found in the available animal studies. The most conservative model for predicting cancer rates was also used. The worst case analysis for synergistic effects assumed that these effects could occur. The probability of these effects occurring was considered low.

EPA has identified the data gaps shown in Table 1-1 in accordance with the registration guidelines under the Federal Insecticide, Fungicide, and Rodenticide Act. Although there are data gaps or areas of uncertainty for some of the herbicides in this risk assessment, there is a large body of existing data that is useful in predicting the behavior and toxicity of these herbicides. These studies include the following:

1. Worker exposure studies with 2,4-D, 2,4-DP, dicamba, amitrole, and picloram.
2. Studies on drift of 2,4-D and glyphosate.
3. Residue information for a number of the herbicides in plant and animal tissues.
4. Cancer studies for those herbicides without mutagenicity studies.
5. Chronic feeding studies that provide some evidence of cancer.
6. Studies either not reviewed by EPA, or validated studies reviewed by EPA, but determined not to be adequate to meet current registration standards, which nonetheless provide some information on toxic effects.

ORGANIZATION OF THIS SUPPLEMENT

Section 1 presents the purpose, describes the structure, and outlines the methodology of the risk assessment. Section 2 outlines the vegetation management programs that use herbicides and the mitigation measures practiced in each. Section 3, the hazard analysis, summarizes and discusses the toxic properties of each herbicide, including the cancer potency of the known or suspected carcinogenic herbicides. Section 4, the exposure analysis, describes the methods used to estimate levels of exposure and resultant doses to workers and the public and presents summary tables and discussions of estimated acute and long-term doses. Section 5, the risk analysis, presents the comparison of the results of the exposure analysis with the toxic effect levels set forth in Section 3. Section 5 also discusses cancer risk, given estimated lifetime doses to workers and the public. Attachment A presents the details of the mutagenicity and cancer testing done on many of the herbicides. Attachment B provides the complete dose estimates for workers and the public derived from the methods described in the exposure analysis. Attachment C presents the complete margin-of-safety tables used in the risk analysis.

Appendix D
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Section 2

Section 2

VEGETATION MANAGEMENT PROGRAMS

This section describes the vegetation management programs that the Forest Service and BLM conduct in Washington and Oregon involving the use of herbicides. The first subsection briefly describes the different types of vegetation management programs that use herbicides. The second subsection identifies the application methods and the principal herbicides used in those programs. The final subsection discusses mitigation measures used to minimize the possible adverse effects of the herbicides on human health and the environment. Complete descriptions of the Forest Service and BLM vegetation management programs are found in the environmental impact statements that this document supplements.

PROGRAM DESCRIPTIONS

The Forest Service and BLM conduct vegetation management programs on Federal lands in Washington and Oregon to sustain and improve the ability of those lands to produce timber, livestock forage, and wildlife; to ensure public safety on roads, other rights-of-way, and recreation sites; and to protect facilities and capital improvements. Herbicides are proposed for use in these programs as described in the Forest Service and BLM environmental impact statements cited in Section 1.

Silviculture operations, designed to ensure the establishment and healthy growth of timber crop species, are the largest proposed program for herbicide treatment by both the Forest Service and BLM (see Table 2-1). These operations include site preparation, plantation maintenance, conifer release, precommercial thinning, and noncommercial tree removal. Site preparation treatments are used to prepare newly harvested or inadequately stocked areas for planting a new crop of trees. Use of herbicides in site preparation reduces vegetation that would compete with the conifers. In the brown and burn method of site preparation, herbicides are used to dry the vegetation, which several months later is burned. Herbicides are used in plantations some time after planting to promote the survival and establishment of conifers (maintenance) or to promote the dominance and growth of already established conifers (release). Precommercial thinning reduces competition among conifers, thereby improving the growth rate of the crop trees. Noncommercial tree removal is used to eliminate dwarf mistletoe-infected host trees. These latter two silvicultural practices primarily use manual methods, although the use of herbicides constitutes about 2 to 5 percent of the operations. On the basis of total acreage managed, the Forest Service has historically used herbicides in about 12 percent of its site preparation work, BLM in about 30 percent. The Forest Service has

Table 2-1

Typical Annual Acres Treated by Vegetation Management Program

Application Method	Silviculture	Noxious Weeds	Rights-of-Way	Facility Maintenance	Range Management	Total
<u>Forest Service, Region 6</u>						
Aerial Treatments	16,500	100	300	0	1,750	18,650
Ground Treatments	<u>14,500</u>	<u>1,400</u>	<u>4,200</u>	<u>125</u>	<u>1,750</u>	<u>21,975</u>
All Treatments	31,000	1,500	4,500	125	3,500	40,625
<u>Bureau of Land Management, Western Oregon</u>						
Aerial Treatments	34,500	0	400	0	-	34,900
Ground Treatments	<u>7,000</u>	<u>275</u>	<u>1,500</u>	<u>25</u>	<u>-</u>	<u>8,800</u>
All Treatments	41,500	275	1,900	25	-	43,700

used herbicides in approximately 80 percent of its maintenance and release projects, BLM in more than 90 percent.

Right-of-way management operations include roadside maintenance and maintenance of power transmission lines, waterways, and railroad corridors. In roadside maintenance, vegetation is removed from ditches and the shoulders of roads to prevent brush encroachment into driving lanes, to maintain visibility on curves for the safety of vehicle operators, to permit drainage structures to function as intended, and to facilitate maintenance operations. Herbicides have been used in 16 percent of the Forest Service's roadside maintenance in Region 6. In western Oregon, 30 percent of BLM's roadside maintenance has historically used herbicides.

Noxious weed control programs control noxious and poisonous plants harmful to humans or domestic livestock. Plants most often treated are poison oak,

ransy-ragwort, St. Johnswort, skeleton weed, and thistle. BLM's noxious weed control program is analyzed in a separate EIS, "Northwest Area Noxious Weed Control Program" (BLM, 1985).

The Forest Service and BLM have used herbicides extensively in their noxious weed program. The Forest Service has historically used herbicides on almost all acres of noxious weed treated.

Range improvement operations provide forage for domestic livestock grazing by removing undesirable competing plant species and preparing seedbeds for desirable plants. The Forest Service uses herbicides on about 12 percent of its range improvement acreage in Region 6. BLM's range improvement program is evaluated in a separate EIS (BLM, 1985).

Facilities and recreation site maintenance operations provide for the safe and efficient use of Forest Service and BLM facilities and recreation sites and for permittee/grantee use of such public amenities as ski runs, waterways, and utility terminals. BLM includes its facility maintenance in its roadside maintenance and weed control program. The Forest Service uses herbicides on less than 11 percent of the total acreage maintained by its facility and recreation site maintenance operations.

APPLICATION METHODS AND HERBICIDE USAGE

Herbicides are applied either from the air or on the ground. Aerial methods employ boom-mounted nozzles carried by helicopters or fixed-wing aircraft. Ground application methods include vehicle-mounted, backpack, and hand application techniques. Vehicle-mounted application systems use fixed-boom or hand-held spray nozzles mounted on trucks or tractors. Backpack systems use either a pressurized sprayer or a powered mist blower to apply herbicides as a broadcast spray directly to one or a group of individual plants.

The principal hand application techniques are injection and stump treatment. Injection involves the application of herbicide in hand-held containers or injectors through slits cut into the stems of target plants. Individual stem treatment by the injection method also is used for crop tree thinning or removal of weed trees. Hack-and-squirt and injection bar equipment are most often used in injection treatments. Stump treatment entails directly applying liquid herbicide to the cut stump of the target plant. The herbicide can be applied by dabbing or painting the stump, or using a squeeze bottle on a freshly cut surface to inhibit sprouting. Herbicides may also be applied by hand in solid form as granules spread on the ground surface.

Although all of the application methods have been used in every type of management operation (except aerial methods on facility or recreation sites), only one or two methods are routinely used. Table 2-2 lists the application methods used in each type of management program with an indication of which methods are commonly used and which are only rarely used. Table 2-1 lists Forest Service and BLM acres treated in each management program by aerial and ground methods in a typical year. Actual historical data were used in determining these typical acreages.

The principal herbicides used by both agencies in terms of total acres treated in all programs are 2,4-D, glyphosate, and triclopyr. Figures 2-1 and 2-2 illustrate the historical proportion of total treated acreage for each of the 16 herbicides used by the Forest Service and BLM.

Aerial Methods

The Forest Service treats more than half of its herbicide-treated silviculture and range management sites by air, as indicated in Table 2-1. BLM treats more than 80 percent of its silviculture sites by air. In general, helicopters are used on silviculture projects because the many treatment units are far apart, small and irregularly shaped, and in steep terrain. Herbicides are normally released 30 to 90 feet above vegetation as medium-sized droplets in an 80- to 90-foot swath. On an average day, several treatment units totaling 150 acres can be sprayed.

Fixed-wing aircraft commonly are used on range management and noxious weed projects in which large contiguous areas are treated. Herbicides are generally released at the same height and swath width as in helicopter treatments. For a large treatment unit, 400 acres can be treated each day.

Batch trucks are an integral part of any aerial operation. They serve as mixing tanks for preparing the correct proportions of herbicide and carrier, and they move with the operation when different landing areas are required.

The number of workers involved in a typical aerial spray project varies according to the type of activity. A small operation may require only 6 individuals, while a complex spray operation may need as many as 20 to 25 workers. The aerial operations crew for range management, noxious weed control, and right-of-way maintenance normally consists of five to eight individuals. Typical personnel on a large project include a pilot, a mixer-loader, a contracting officer's representative (COR), an observer-inspector, a one to six-member card crew, one or two law enforcement officers, one or two water monitors, and one or two laborers. Optional personnel include an air operations officer, a radio technician, a weather monitor, and a recorder.

The following discussions are based on historical data on actual acres treated from the Forest Service Region 6 and BLM in western Oregon.

Forest Service Aerial Projects

In terms of total annual Forest Service herbicide use, the aerial application of herbicides in silviculture and range management programs normally constitutes about one-third of the herbicide applied.

2,4-D, glyphosate, and triclopyr have historically been the principal herbicides used for the Forest Service's aerial silviculture operations. The main herbicides used in aerial range management have been atrazine, dalapon, and 2,4-D. Picloram and 2,4-D have been the principal herbicides used on the small number of acres treated aerially for noxious weed control and right-of-way maintenance by the Forest Service. In some years, there has been no aerial spraying of rights-of-way.

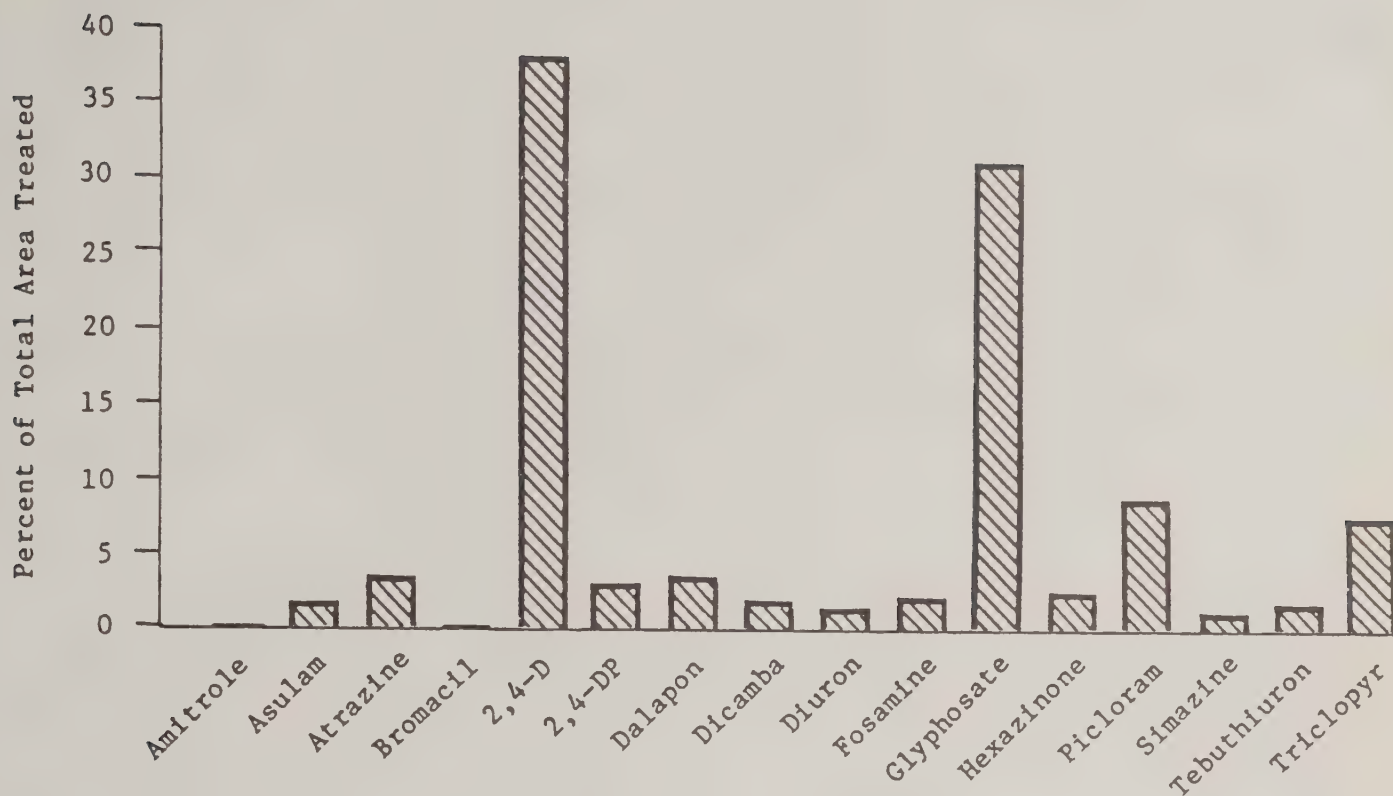


Figure 2-1 Historical Herbicide Use in Region 6 National Forests

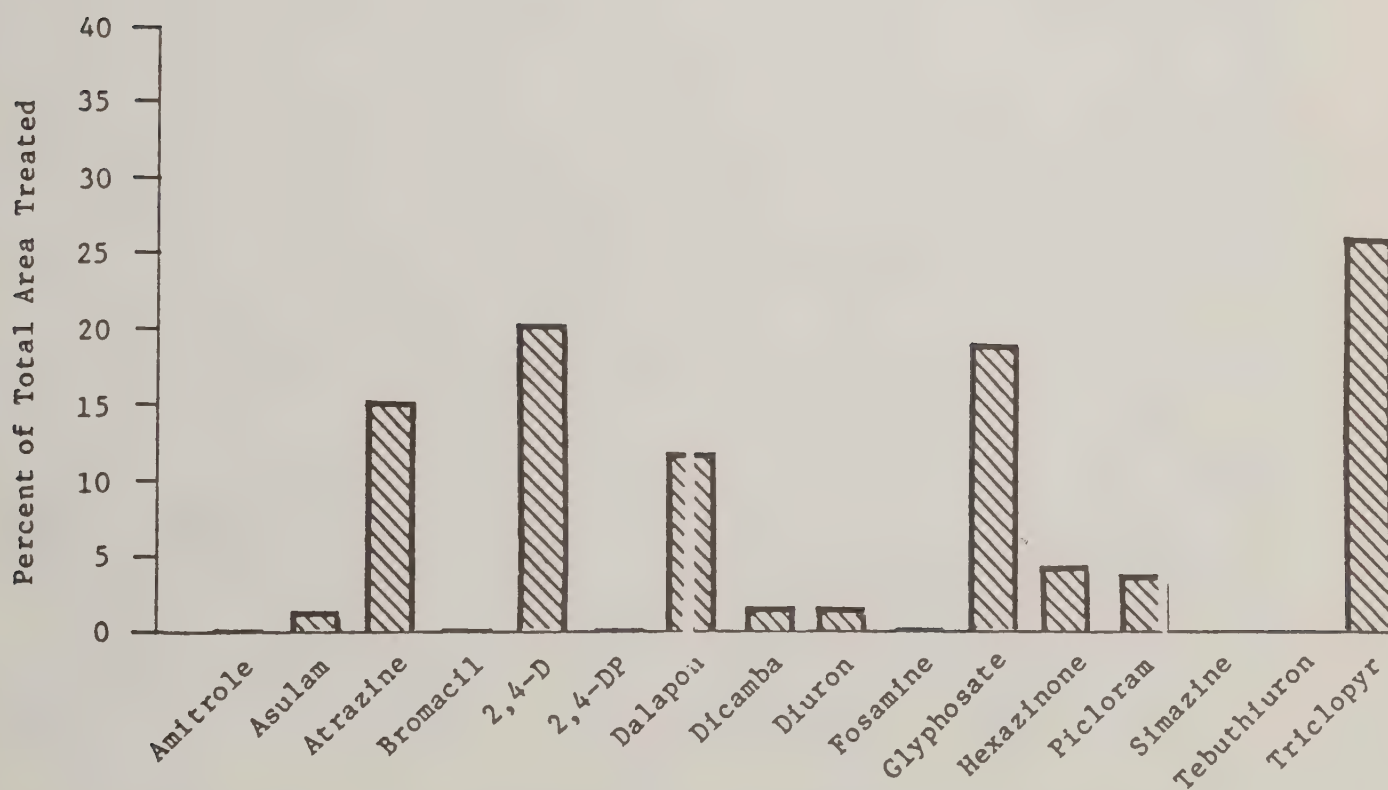


Figure 2-2 Historical Herbicide Use by BLM in Western Oregon

Aerial silviculture treatment units vary in size from 2 acres to 60 acres. Normally, aerial treatment units are no more than 40 acres. Based on a 150-acre-per-day application schedule, there were roughly 100 total treatment days. Region 6 Forest Service personnel estimate that aerial silviculture programs require 200 to 250 total workers and 2,500 to 3,500 total worker days of labor each year. Range improvement operations may include two or three large aerial projects per year, with treatment units ranging up to 400 acres. The annual work force for range projects was estimated at 25 to 30 workers and 300 to 350 total worker days.

BLM Aerial Projects

Historically, more than 80 percent of the total acres treated by BLM was for aerial silviculture projects. Five principal herbicides have historically been used by BLM aerial silviculture programs: 2,4-D, glyphosate, triclopyr, atrazine, and dalapon. Aerial treatments for right-of-way maintenance using primarily 2,4-D and triclopyr normally accounted for less than 1 percent of the total treated acreage.

Silviculture projects make up the bulk of BLM's aerial operations. BLM generally applies herbicides on about 150 acres/day in aerial treatments. An average of six individuals are normally involved in each of BLM's aerial spray operations.

Ground-Based Methods

Forest Service Ground-Based Treatment Projects

Silviculture and Range Management Projects. Ground treatment in silviculture and range management programs accounted for nearly half of the total acreage treated with herbicides by the Forest Service in 1982 and 1983. Glyphosate, 2,4-D, picloram, and triclopyr were the major herbicides used during ground-based silvicultural treatment in those years. Rangeland was treated predominantly with atrazine, dalapon, and 2,4-D.

Backpack treatment is the predominant ground-based method used for silviculture, although stump treatment and injection also are used. Herbicides may also be applied in granular form. Backpack treatment is also the predominant ground-based method used in range management.

Pressurized backpack treatment operations typically involve a supervisor (who may also function as a mixer-loader), an inspector, a monitor, and 2 to 12 crew members. Backpack sprayers can typically treat one-half of an acre per hour in silviculture operations. Four laborers and one inspector generally make up the work force for stump treatment or injection.

Major ground-based silviculture programs of the Forest Service in 1982 involved treatment of 300 or more acres in eight of the National Forests within Region 6. A total of 100 to 150 workers and 3,800 to 4,300 total worker days was estimated as a yearly labor allotment.

Right-of-Way Projects. In Region 6, areas treated with herbicides during right-of-way and road maintenance projects historically account for about one-fifth of the total acres treated. Treatments are normally done by permittees of the Forest Service (State highway departments, county road crews, utility companies, and the like). Fosamine, 2,4-D, 2,4-DP, picloram, tebuthiuron, and diuron were the most heavily used herbicides in this program.

Right-of-way maintenance projects frequently use vehicle-mounted application techniques. A truck with a mixing/holding tank uses a front-mounted spray boom or a hand-held pressurized nozzle to treat roadside vegetation on varying slopes. Use of this equipment for off-road right-of-way projects is limited to gentle slopes (less than 20 percent) and open terrain. Contractors spray an average of 30 to 50 acres per day with vehicle-mounted applicators. A driver/mixer-loader and applicator constitute the typical crew for truck spraying. A total of 3,000 to 3,500 worker days and 100 to 125 workers was estimated as a yearly quota for right-of-way maintenance projects.

Noxious Weed Control Projects. Forest Service use of ground-applied herbicides for noxious weed control normally accounted for less than 5 percent of the total acreage treated in both 1982 and 1983. Nearly half of the noxious weeds affected were on rangelands. 2,4-D, picloram, and dicamba are the principal herbicides used for noxious weed control. Backpacks, spray bottles, and trucks or tractors with spray booms or tractor-mounted attachments are used in ground-based noxious weed programs. Backpack sprayers can typically treat only 1 acre every 3 to 4 hours in noxious weed control programs because target plants are normally found as scattered individuals or in small groups. About one-fourth of the total acres treated in noxious weed control projects was hand-treated with herbicides in granular form. Noxious weed control programs, using both aerial and ground methods, account for 1,500 to 2,200 total worker days and 140 to 150 workers per year.

Facility Maintenance Projects. Facility maintenance by the Forest Service resulted in treatment of about 1 percent of the total acreage controlled by herbicides in 1982 and 1983. Amitrole, glyphosate, 2,4-D, and 2,4-DP were the major herbicides used in 1982, while glyphosate and 2,4-D were the predominant herbicides applied in 1983. All methods of ground application may be used and would typically involve only one or two applicators and one supervisor who would check on the work after the task was completed. Many small short-term projects throughout the Region have resulted in a total treatment of 100 to 125 acres annually.

BLM Ground Application Projects

Ground-based methods of herbicide application are not used as extensively by BLM as they are by the Forest Service. Manual methods are often used in silviculture projects, and controlled burning is commonly used for site preparation. In silviculture projects, ground applications normally constitute less than 20 percent of the total area treated by BLM.

Methods of herbicide application in BLM ground-based operations are similar to those of the Forest Service. Ground application in BLM projects is accomplished through backpack spraying, vehicle-mounted spraying, injection, stump treatment, and other hand application methods.

Triclopyr, glyphosate, atrazine, and dalapon accounted for more than 95 percent of the total herbicides chosen for site preparation operations under the proposed alternative of the EIS for the Western Oregon Program--Management of Competing Vegetation (1983). These four herbicides and 2,4-D accounted for nearly 90 percent of the herbicides selected for use in the maintenance and release projects under the proposed alternative for BLM's silviculture program. BLM's right-of-way maintenance projects used triclopyr, 2,4-D, dicamba, and diuron for almost all of the acres treated. BLM's ground spraying projects are about the same size as the Forest Service's.

MITIGATION MEASURES

Mitigation measures are intended to ensure the proper and safe application of herbicides on Forest Service and BLM lands in Washington and Oregon and are required by Federal, State, and regional procedures. Federal and State laws and regulations set minimum standards to be followed during herbicide application on forests and rangelands owned by the Federal Government. Each regional and district office also may develop additional restrictions and precautions. The Federal Insecticide, Fungicide, and Rodenticide Act requires that pesticide manufacturers register their chemicals with the U.S. Government and list the allowable uses, application rates, and special restrictions on the herbicide's label. All of the herbicides considered in this risk assessment are registered with the Environmental Protection Agency; and their label rates, uses, and handling instructions must be complied with according to Federal law.

The Department of the Interior (Bureau of Land Management) and the Department of Agriculture (Forest Service) have handbooks that prescribe guidelines for aerial and ground application operations. Regional publications, such as BLM's Western Oregon Program--Management of Competing Vegetation Environmental Impact Statement and the Forest Service's Region 6 Vegetation Management Program Environmental Impact Statement, serve to further refine herbicide application guidelines. The Siskiyou National Forest Aerial Applicator's Handbook (USDA, 1982) is an example of a forest level operational guideline that specifies detailed herbicide application procedures.

Aerial and ground application procedures undergo detailed planning weeks or even months in advance. Mitigation measures, such as not spraying in sensitive areas, notifying the public, posting warning signs, and conducting water monitoring, are specified in site-specific annual vegetation management plans.

Many mitigation measures developed for herbicide operations in Washington and Oregon are described in each agency's environmental impact statements, which this document supplements. Some specific examples of project mitigation measures include the following:

1. Application operations will be suspended when any of the following conditions exist:
 - a. Wind velocity exceeds 5 miles per hour
 - b. Air temperature exceeds 70 °F
 - c. Relative humidity is less than 50 percent
 - d. It is raining or misting or there is a 40-percent chance of rain within several hours
 - e. Foggy weather
2. During air operations, a radio network will be maintained to link all parts of the project.
3. Equipment is designed to deliver a median droplet diameter of 200 to 800 microns. This droplet size is large enough to avoid excessive drift while providing adequate coverage of target vegetation.
4. Individuals involved in the herbicide handling or application will be instructed on the safety plan and spill procedures.

Table 2-2

Herbicide Application Methods Used in Forest Service
and Bureau of Land Management Vegetation Management Programs

Application Method	Project Type					
	Silviculture			Facilities		
	Site Preparation	Conifer Release	Range Improvement	Noxious Weeds	Right-of-Way Maintenance	and Recreation Site Maintenance
<u>Aerial</u>						
Fixed Wing	R	R	O	O	R	
Helicopter	C	C	O	O	C	
<u>Mechanical</u>						
(Truck- mounted or towed sprayer)	R	R	O	O	C	O
<u>Backpack</u>						
Backpack	C	C	O	O	O	C
<u>Hand</u>						
Hand	O	O	R	R	O	O

Legend

C = Commonly Used
 O = Occasionally Used
 R = Rarely Used
 Blank = never used

Appendix D
Human Health Risk
Assessment
(Quantitative)

Section 3

Section 3

HAZARD ANALYSIS

INTRODUCTION

This section presents the results of the hazard analysis: a review of available toxicological information on the 16 herbicides--amitrole, asulam, atrazine, bromacil, 2,4-D, 2,4-DP, dalapon, dicamba, diuron, fosamine, glyphosate, hexazinone, picloram, simazine, tebuthiuron, and triclopyr--that are to be used in the Forest Service and BLM vegetation management programs in the Pacific Northwest. The first subsection describes the sources of toxicity information used in the hazard analysis. The second subsection explains the terminology concerning laboratory toxicity testing used later in describing the toxic properties of the 16 herbicides. The third subsection presents summaries of the threshold toxicity of each herbicide drawn from the information that was available. The fourth and fifth subsections describe the potential for each of the 16 herbicides to cause the nonthreshold effects of genetic mutations and cancer, respectively. The final subsection presents the details of the derivation of cancer potency for those herbicides suspected of being carcinogenic.

SOURCES OF TOXICITY INFORMATION

The toxicity of 12 of the herbicides (amitrole, atrazine, 2,4-D, 2,4-DP, dalapon, dicamba, fosamine, glyphosate, hexazinone, picloram, simazine, and triclopyr) to both laboratory animals and humans is described in detail in the background statements of the Forest Service Agricultural Handbook No. 633 (USDA, 1984). Tebuthiuron toxicity is described in a background statement prepared for the Forest Service as a supplement to Handbook No. 633. The toxicity of the herbicides asulam, diuron, and bromacil is described in background statements written in conjunction with this risk assessment. These documents are incorporated by reference into this Supplement to the Final Forest Service and BLM EIS's identified in Section 1 in accordance with 40 CFR 1502.16 and are available for review at all Forest Service and BLM District Offices in Oregon and Washington, as well as at the address shown on the cover page.

Much of the data on pesticide toxicity have been generated to comply with the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), as amended (7 U.S.C. 136 et seq), which establishes procedures for the registration, classification, and regulation of all pesticides, including herbicides. EPA is responsible for implementing FIFRA. Toxicity levels and related information from the series of studies submitted for registration are compiled by EPA in summary tables called "tox one-liners" that are available on request from EPA's Freedom of Information Office. EPA has compiled "science chapters" on many of the herbicides (amitrole, bromacil, dicamba, diuron, hexazinone, picloram, and simazine) and these are also available from EPA. A large body of additional toxicity information exists in the open literature, particularly for chemicals such as 2,4-D that have been used for many years.

An extensive literature search was funded by the U.S. Department of Agriculture, Forest Service, to ensure that all of the relevant available information was used in this risk analysis. The Medline data base, the Embase (Excerpta Medica) data base, and the International Pharmaceutical Abstract data base were searched to locate current literature pertaining to the carcinogenicity and mutagenicity of the herbicides.

The data from the U.S. Department of Agriculture, Forest Service, Pesticide Background Statements (1984) were reviewed and compared to summaries of studies submitted to the Environmental Protection Agency for the registration of the 16 herbicides. Whenever possible, studies that have been reviewed and validated by EPA were used to set toxicity reference levels. In no cases were studies used that have been invalidated by EPA.

HAZARD ANALYSIS TERMINOLOGY

Because of obvious limitations on the testing of chemicals on humans, judgments about the potential hazards of pesticides to humans are necessarily based on the results of toxicity tests on laboratory animals. These toxicity test results are supplemented by information on actual human poisoning incidents and effects on human populations when they are available. The discussion of laboratory toxicity testing that follows is drawn from Hayes (1982), Doull et al. (1980), and Loomis (1978).

Laboratory Toxicity Testing

Test Animal Species

Laboratory test animals function as models of the likely effects of the pesticide in humans. Ideally, the test animal should metabolize the compound the same as a human would and should have the same susceptible organ systems. Results of such tests then can be directly extrapolated to humans with some adjustment made for differences in body weight and body surface area. Although no test animal has proven ideal, a number of species have proven to be consistent indicators for certain types of toxicity tests, routes of administration, and types of chemicals; in particular, rats, mice, rabbits, hamsters, guinea pigs, dogs, and monkeys.

Toxic Endpoints and Toxicity Reference Levels

Toxicity tests are designed to produce specific toxic endpoints, such as fatality or cancer, and toxicity reference levels, such as a no-effect level. In addition to the test animal used, toxicity tests vary according to test duration, route of administration, dose levels, dosing schedule, number of test groups, and number of animals per group. Toxicity tests also vary on the basis of whether it is assumed that the effect in question is a threshold effect or a nonthreshold effect.

Threshold and Nonthreshold Effects

Most chemicals are assumed to have a threshold level of toxic effects on a local basis (at the site of administration) or systemic basis (acting throughout the body), below which no adverse effects occur to the test organism. Chemicals are generally thought to possess no such threshold level for cancer and mutations, thus these toxic endpoints may occur (with a certain level of probability) even in the presence of extremely small quantities of the substance. In this hazard analysis, threshold effects are discussed first, in the following subsection. The nonthreshold effects, mutagenicity and cancer, are discussed in the last two subsections.

Duration of Toxicity Tests

The duration of toxicity tests ranges from very short-term acute tests to longer subchronic studies to chronic studies that may last the lifetime of an animal. Acute toxicity studies involve administration of a single dose to each member of a test group (either at one time or in a cumulative series over a short period of less than 24 hours). Subchronic toxicity studies, used to determine the effects of multiple doses, usually last from a few days to 3 months (3 to 90 days) but generally less than one-half the lifetime of the test animal. Chronic studies, also used to determine the effects of multiple or continuous doses, normally last 2 years but generally more than one-half the test species' lifetime.

Routes of Administration

Routes of administration include oral via gavage (forced into the stomach with a syringe through a plastic tube) or fed in the diet, dermal (applied to the skin), inhalation (through exposure to vapors or aerosol particles), and parenteral (injection other than into the intestine) routes, such as subcutaneous (SC), which means injected under the skin, intraperitoneal (IP), meaning injected into the abdominal cavity, and intravenous (IV), meaning injected into a vein. Oral, dermal, and inhalation doses most nearly duplicate the likely routes of exposure to humans. Parenteral doses are used in testing drugs but are not widely used in toxicity testing of pesticides because they bypass the test animal's natural protective mechanisms. Doses are expressed in several ways. They can be expressed as milligrams (mg, which is 1/1,000 of a gram) of the chemical per kilogram (kg, which is 1,000 grams) of body weight of the test animal, or in parts per million (ppm) in the animal's diet, or in milligrams per liter (mg/L) in the air the animal breathes.

Dosing Levels

Dosing in longer term studies is generally done through the diet with specified amounts in parts per million in the food. The known weight of the test animals over the test period is used to convert ppm in the diet to milligrams of chemical per kilogram of body weight per day (mg/kg/day) for extrapolation to humans. Generally, at least three dosing levels are used in addition to a zero dose or control group. Animals of each sex usually are dosed in groups of 8 to 50.

Types of Toxicity Studies

Acute Toxicity Studies

Acute toxicity studies are used to determine the toxicity reference level known as the median lethal dose (LD₅₀), which is the dose that kills 50 percent of the test animals. The lower the LD₅₀, the greater the toxicity of the chemical. The LD₅₀ ranges and toxicity categories used in this risk assessment are those of the EPA classification system using rat LD₅₀'s, as shown in Table 3-1 (adapted from Walstad and Dost, 1984). Because lethality is the intended toxic endpoint, dose levels usually are set relatively high in acute studies. Toxic symptoms displayed by the animals may be recorded throughout the study, and tissues and organs are examined for abnormalities at the end of the test. The animal most commonly used for oral LD₅₀'s is the rat. Rabbits are used most often to determine dermal LD₅₀'s.

(insert Table 3-1)

Because death represents the extreme toxic consequence for judging possible effects from the use of pesticides, the policies of regulating agencies regarding acceptable intake levels of these chemical compounds are most often based not on acute studies, but rather on toxicity tests designed to find the dose level that produces no effects in the animal species tested. Figure 3-1 illustrates the relationship between the LD₅₀ and the no-effect level.

Subchronic Toxicity Studies

Subchronic studies are designed to determine the toxicity reference level called the no-observed-effect level (NOEL), which is the highest dose level at which no toxic effects are observed. If a chemical produces effects at the lowest dose tested (LDT) in a study, the NOEL must be at some lower dose. If the chemical produces no effects, even at the highest dose tested (HDT), the NOEL is equal to or greater than the HDT. Another toxic endpoint of interest is the lowest dose showing toxic effects, the lowest effect level (LEL). For local and systemic effects, the chemical's threshold of effect lies between the NOEL and LEL for the tested species (see Figure 3-1).

Subchronic studies, normally employing lower dose levels than acute studies, provide information on systemic effects, cumulative toxicity, the latency period (the time between exposure and the manifestation of a toxic effect), the reversibility of toxic effects, and appropriate dose ranges to be used in chronic tests. The adverse effects may include death; decreased rate of food consumption; change in body weight; decreased enzyme levels; changes in blood constituents, such as red blood cells (RBC's) or white blood cells (WBC's); undesirable constituents in the urine; or microscopic changes in tissues.

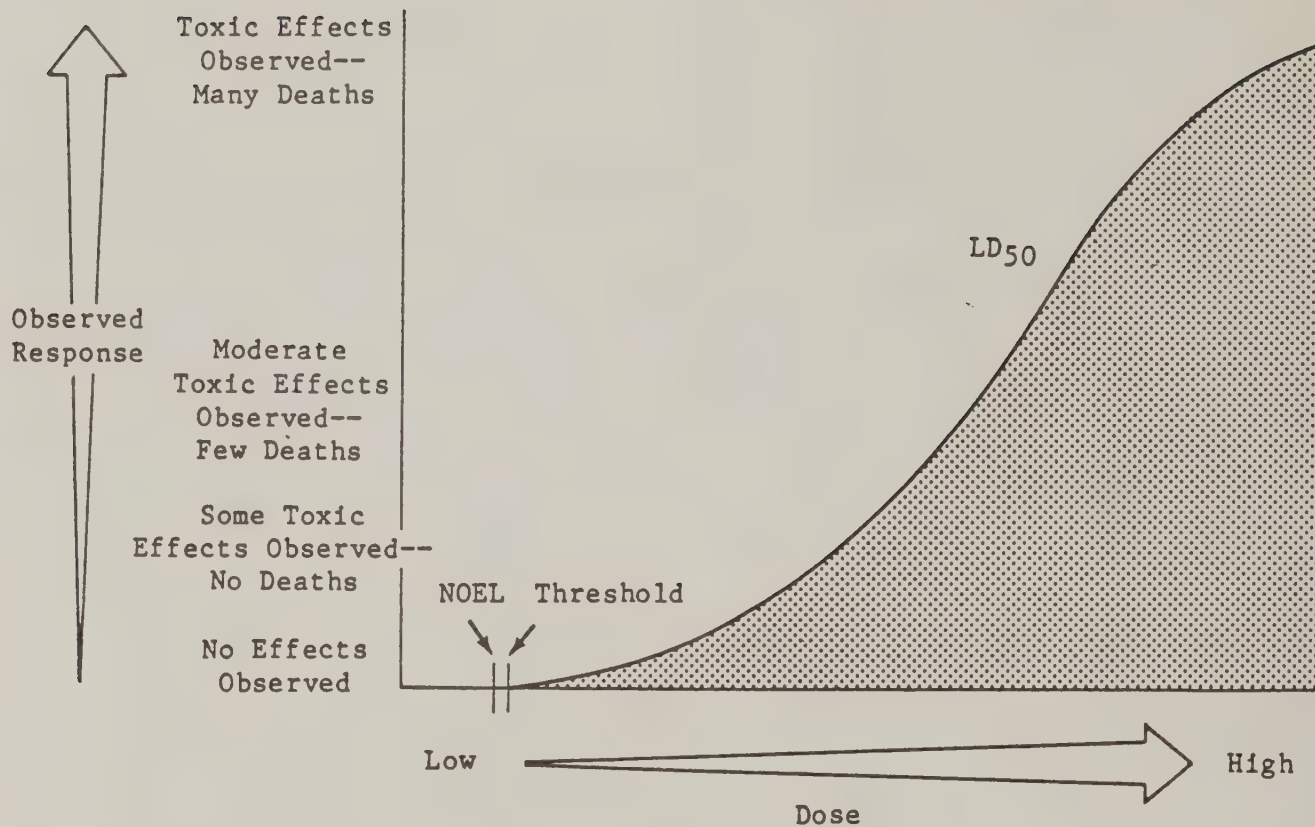
Table 3-1

Acute Toxicity Classification and Acute Toxicities of
the 16 Herbicides and Other Chemicals

Toxicity Category ¹ (label signal words)	Herbicide or Other Chemical Substance	Oral LD ₅₀ for Rats (mg/kg)	Equivalent Human Dose
IV Very slight		5,000 - 50,000 (range)	More than 1 pint
	Sugar	30,000	
	Fosamine	24,400	
	Ethyl alcohol	13,700	
	Picloram	8,200	
	Dalapon	7,577	
III Slight (caution)		500 - 5,000 (range)	1 ounce to 1 pint
	Simazine	5,000	
	Glyphosate	4,320	
	Amitrole	4,080	
	Asulam	4,060	
	Bromacil	3,998	
	Table salt, diuron	3,750	
	Bleach	2,000	
	Atrazine	1,869	
	Aspirin, Vitamin B ₃	1,700	
	Hexazinone	1,690	
	Dicamba	757	
	Tebuthiuron	644	
	Triclopyr	630	
	2,4-DP	532	
II Moderate (warning)		50 - 500 (range)	1 teaspoon to 1 ounce
	2,4,5-T	500	
	2,4-D	375	
	Caffeine	200	
	DDT	100	
I Severe (danger - poison)		0 - 50 (range)	1 teaspoon or less
	Nicotine	50	
	Strychnine	30	
	(rodenticide)		
	Parathion	13	
	(insecticide)		
	TCDD (dioxin)	0.001	
	Botulinus Toxin	0.00001	

¹Categories, signal words, and LD₅₀ ranges are based on a classification system used by the EPA for labeling pesticides.

Source: Maxwell, 1982 (as cited in Walstad and Dost, 1984).



LD₅₀ - Acute lethal dose. One-time or short-term dose that is lethal to 50 percent of treated animals.

Threshold - Dose level at which toxic effects are first observed in test animals.

NOEL - No-observed-effect level. Long-term dose that does not result in apparent adverse effects in test animals.

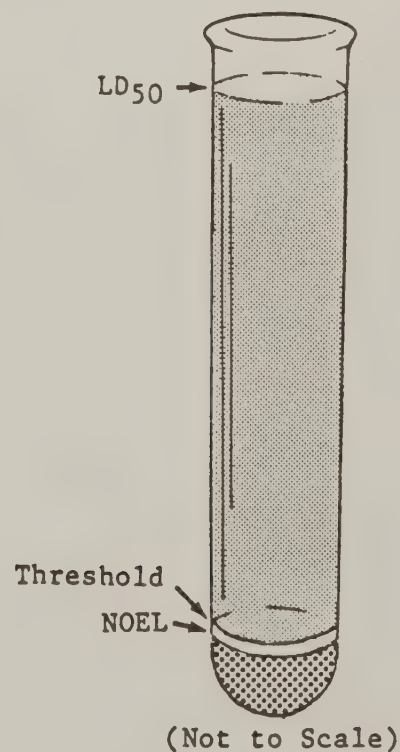


Figure 3-1 Relationships Among Toxicity Reference Levels

Teratogenicity tests. Teratogenicity tests (teratology studies) determine the potential of a chemical to cause malformations in an embryo or a developing fetus between the time of conception and birth. These studies generally use pregnant female rats or rabbits dosed during the middle period of gestation while the organs of the fetus are developing. The animals are monitored for functional as well as structural deformities.

Chronic Toxicity Studies

Chronic studies, like subchronic studies, can be used to determine systemic NOEL's. All other things being equal, the longer the study from which the NOEL is derived, the more reliable the resulting value. Chronic studies, however, are even more important in determining doses that are hazardous to reproductive success or in determining whether the chemical causes cancer. Chronic tests include feeding studies, reproduction studies, and carcinogenicity tests.

Reproduction studies. Reproduction studies are conducted to determine the effect of the chemical on reproductive success as indicated by fertility direct toxicity to the developing fetus, and survival and weight of offspring for low-level, long-term exposure. These tests are usually performed at lower doses than those used in teratogenicity studies and generally use rats. Both male and female rats are exposed to the chemical for a number of weeks before mating. The number of resulting pregnancies, stillbirths, and live births are recorded. Tests may be conducted over two or three generations.

Carcinogenicity tests. Carcinogenicity tests (cancer studies or oncogenicity studies) examine the potential for a chemical to cause tumors when fed in the diet over the animal's lifetime. Testing is normally conducted with rats or mice for a 2-year period.

Mutagenicity Assays

Mutagenicity assays are used to determine the ability of a chemical to cause physical changes (mutations) in the basic genetic material (DNA) that could be passed on from one generation to the next. The species used in these tests range from primitive organisms, such as the bacteria Salmonella, Escherichia, and Streptomyces; the mold Aspergillus; the yeast Saccharomyces; and the fruit fly Drosophila, to the more advanced organisms that include mammalian species. Tests may be conducted in vivo (within the body of the living organism) or in vitro (on cells cultured outside the body in a petri dish or test tube). There are many types of tests in this category, including the dominant lethal assay, which is usually conducted with rodents, and the Ames reverse mutation test, which is conducted with bacteria. The sub-section on mutagenicity of the 16 herbicides gives more information about mutagenicity tests.

THRESHOLD TOXICITY OF THE 16 HERBICIDES

The toxicity reference levels used in this risk assessment to describe both acute and chronic threshold effects of the 16 herbicides are presented in Table 3-2. The LD₅₀'s in this table are from rat oral studies. Two types of NOEL's are given in Table 3-2. The first NOEL is for general systemic effects, such as growth retardation, decreased red blood cell counts, and increased thyroid weight. All of the systemic NOEL's take into account validated 2-year chronic feeding studies. For amitrole, asulam, fosamine, picloram, tebuthiuron, and triclopyr, subchronic study NOEL's were used because they are the lowest NOEL's found in the literature. The second NOEL is for reproductive effects, including infertility, miscarriage, general fetal toxicity, and birth defects (teratogenesis). Where information is available, NOEL's are given for both reproductive and teratogenic effects. All the NOEL's used are the lowest found in validated studies.

(insert Table 3-2)

The following subsections summarize the most relevant acute and chronic toxicity tests that have been conducted on 16 herbicides. Areas where no validated studies exist or in which EPA has requested additional studies are noted.

Amitrole

Amitrole can be classified as slightly toxic (see Table 3-1) based on the acute oral LD₅₀ value in rats of greater than 4,080 mg/kg (EPA, 1984a). The lowest systemic NOEL found in the literature, based on thyroid effects in a subchronic feeding study with rats, was reported to be 0.5 ppm (0.025 mg/kg/day) (EPA, 1985a).

EPA's risk assessment of amitrole (EPA, 1985a) states the following:

In a subchronic feeding study (Fregly, 1968), male rats were fed 0, 0.25, 0.50, 2.00, 10, and 50 ppm amitrole in the diet for 11 or 13 weeks. Thyroid iodine uptake was reduced, and enlarged thyroids were observed in animals fed 2.00, 10, and 50 ppm. Significant functional changes had already occurred after only 1 week of feeding.

In addition, another subchronic feeding study (Food Drug Research Labs, 1977) fed male rats 0, 30, 100, and 300 ppm amitrole in the diet for 4 weeks followed by 4 weeks on control diet. The study was designed to demonstrate the reversibility of the antithyroid effects of amitrole. Reduced levels of T₃ and T₄ were reported in the 100 and 300 ppm groups after 14 and 7 days, respectively, on the amitrole diets. After 21 days on the control diet during the recovery phase of the study, all treatment groups had T₃ and T₄ values similar to concurrent control values.

In a 2-generation reproduction study, groups of male and female rats (F₀) were fed 500 and 1,000 ppm amitrole for 107 to 110 days. Two other groups were fed 25 and 100 ppm for 240 to 247 days, and their progeny (F₁) were fed 25 and 100 ppm amitrole for 141 days (Gaines, 1973). Pups born to parents fed 500 and 1,000 ppm amitrole were small, and had atopic thymuses and spleens indicative of runting; no signs of runting were observed in the 25 and 100 ppm pups. Hyperplasia of the thyroid was observed in all animals fed 25 ppm and higher.

Table 3-2

Laboratory-Determined Toxicity
Levels Used in the Risk Analysis

Herbicide	Acute Oral LD ₅₀ in Rats	Lowest Systemic NOEL	Lowest Reproductive and/or Teratogenic NOEL
Amitrole	Greater than 4,080 mg/kg (EPA, 1984a)	0.5 ppm (0.025 mg/kg/day) subchronic rat feeding study (EPA, 1985a)	100 ppm (5 mg/kg/day) 2-gen- eration rat reproduction study (EPA, 1985a) No birth defects observed in any studies Fetotoxic NOEL = 5,000 ppm (750 mg/kg/day) mouse teratology study (EPA, 1985a)
Asulam	Greater than 4,000 mg/kg (EPA, 1984b)	50 mg/kg/day 107-week rat feeding study (EPA, 1985d)	1,000 ppm (50 mg/kg/day) 2- generation rat reproduction study (EPA, 1984b) Maternal NOEL = 300 mg/kg, rabbit teratology study (EPA, 1984b) No birth defects observed in any studies
Atrazine	1,869 mg/kg (EPA, 1984c)	150 ppm (3.7 mg/kg/day) 2-year dog feeding study (EPA, 1984c)	No birth defects in two studies Three-generation reproduc- tive NOEL of greater than 100 ppm (50 mg/kg/day), rat (EPA, 1984c) 100 mg/kg/day fetotoxic NOEL rat teratology study (EPA, 1984c)

Table 3-2 (Cont.)

Herbicide	Acute Oral LD ₅₀ in Rats	Lowest Systemic NOEL	Lowest Reproductive and/or Teratogenic NOEL
Bromacil	3,998 mg/kg (EPA, 1984d)	250 ppm (6.25 mg/kg/ day), 2-year dog feeding study (EPA, 1984d)	No teratogenic effects in two studies Greater than 165 mg/m ³ (7.92 mg/kg/day HDT), rat teratology study (EPA, 1984d)
2,4-D	375 mg/kg (EPA, 1984e)	1.0 mg/kg, first year results from 2-year rat feeding study (EPA, 1985c)	Fetotoxic and maternal NOEL = 5 mg/kg/day, rat teratology study (EPA, 1986b)
2,4-DP	532 mg/kg, rat (EPA, 1984f)	50 mg/kg, 2-year rat feeding study (EPA, 1984f) 5 mg/kg 13-week rat feeding study (EPA, 1982)	Three-generation rat reproduction study, NOEL = 125 ppm (6.25 mg/kg/day) (EPA, 1984f) Teratogenic effects at 25 mg/kg/day (LDT). Maternal and fetotoxic NOEL = 25 mg/kg/day, rabbit (EPA, 1984f)
Dalapon	7,577 mg/kg (EPA, 1984g)	15 mg/kg, 2-year rat feeding study (USDA, 1984)	Greater than 300 mg/kg/day, 3-generation rat reproduc- tion (USDA, 1984)

Table 3-2 (Cont.)

Herbicide	Acute Oral LD ₅₀ in Rats	Lowest Systemic NOEL	Lowest Reproductive and/or Teratogenic NOEL
Dicamba	757 mg/kg (USDA, 1984)	500 ppm (25 mg/kg/day) 90-day subchronic feeding study (EPA, 1984h)	No teratogenic effects reported in 4 studies Fetotoxic and maternal NOEL = 3.0 mg/kg/day, rabbit teratology study (EPA, 1984h) Reproductive NOEL = 2.5 mg/kg/day (EPA, 1985d)
Diuron	3,750 mg/kg (EPA, 1984i)	25 ppm (0.625 mg/kg/day) 2-year dog feeding study (EPA, 1984i)	No birth defects. NOEL greater than 125 ppm 3-generation rat reproduc- tion study (6.25 mg/kg/day) (Hodge et al., 1967; EPA, 1984i)
Fosamine	24,400 mg/kg (EPA, 1984j)	1,000 ppm (25 mg/kg/day) 6-month dog feeding study (Schneider and Kaplan, 1983 in USDA, 1984)	Greater than 10,000 ppm (500 mg/kg/day), rat teratology study (Schneider and Kaplan in USDA, 1984)
Glyphosate	4,320 mg/kg (EPA, 1984k)	Greater than 31 mg/kg/day, 26-month rat feeding study (EPA, 1984k)	10 mg/kg, 3-generation reproduction rat study (EPA, 1984k) Maternal NOEL = 175 mg/kg, rabbit teratology study (USDA, 1984). No birth defects

Table 3-2 (Cont.)

Herbicide	Acute Oral LD ₅₀ in Rats	Lowest Systemic NOEL	Lowest Reproductive and/or Teratogenic NOEL
Hexazinone	1,690 mg/kg (EPA, 1984l)	200 ppm (10 mg/kg/day) 2-year rat feeding/ oncogenic study (EPA, 1984l)	1,000 ppm (50 mg/kg/day), 3-generation reproduction rat study (USDA, 1984) 125 mg/kg (HDT), rabbit teratology study (USDA, 1984)
Picloram	8,200 mg/kg, rat	7 mg/kg, 6-month dog feeding study (Mullison, 1985)	No teratogenic effects in 3 studies 1,000 ppm (50 mg/kg/day) rat 3-generation reproduction study (EPA, 1984m)
Simazine	Greater than 5,000 mg/kg (EPA, 1984n)	Greater than 100 ppm (5 mg/kg/day)(HDT), 2-year rat feeding study (EPA, 1984n)	Greater than 100 ppm (5.0 mg/kg/day),(ODT), 3-generation reproduction study (EPA, 1984n)

Table 3-2 (Cont.)

Herbicide	Acute Oral LD ₅₀ in Rats	Lowest Systemic NOEL	Lowest Reproductive and/or Teratogenic NOEL
Tebuthiuron	644 mg/kg (DOE, 1983)	500 ppm (12.5 mg/kg/day), 90-day dog feeding study (EPA, 1975)	No birth defects. Greater than 1,800 ppm (90 mg/kg/ day), reproduction rat study (Todd et al., 1974, in USDA, 1985b) 237 mg/kg, dermal teratology study (EPA, 1984o) Reproductive NOEL greater than 400 ppm (20 mg/kg/day) (HDT), 2-generation repro- duction study with rats (EPA, 1985d)
Triclopyr	630 mg/kg (EPA, 1984p)	2.5 mg/kg/day (HDT), 6-month dog feeding study (40 CFR Part 180, 50 (84):184-85, May 1, 1985)	Fetotoxic NOEL less than 10 mg/kg, rabbit teratology study (EPA, 1984p) No teratogenic effects greater than 30 mg/kg (HDT), 3-generation rat reproduction study (USDA, 1984)

Conversion Factors:

mouse 1 ppm = 0.150 mg/kg/day
 rat (lifetime) 1 ppm = 0.05 mg/kg/day
 rabbit 1 ppm = 0.030 mg/kg/day
 dog 1 ppm = 0.025 mg/kg/day

Source: USDA, 1984

No teratogenic effects were observed at birth through weaning when pregnant rats were orally gavaged from day 7 through 15 of gestation with 20 and 100 mg/kg/day of amitrole (Gaines, 1973). In another teratology study, pregnant mice were treated with 500, 1,000, 2,500, and 5,000 ppm amitrole in the drinking water from day 6 through 18 of gestation (Tjalve, 1974). No teratogenic effects were observed. Indications of fetotoxic effects were observed in the 1,000, 2,500, and 5,000 ppm treatment groups. These effects were small fetuses, underdeveloped fetuses with immature skeletons, and increased number of resorptions (5,000 ppm group only).

The body of nononcogenic data indicates that amitrole has a low acute toxicity in experimental animals. However, amitrole is a potentially potent antithyroid agent as evidenced by the low subchronic feeding levels (2 ppm) that resulted in significant effects on thyroid function. The data also indicate that amitrole does not pose a significant reproductive hazard. However, the data are insufficient to assess teratogenic potential.

EPA has requested additional data to assess the teratogenic potential of amitrole.

Asulam

Based on the acute oral LD₅₀ of greater than 4,000 mg/kg in rats (EPA, 1984b), asulam can be classified as slightly toxic (see Table 3-1). The systemic NOEL of 2,000 ppm (100 mg/kg/day) was based on fatty deposits in the liver derived from a 90-day rat feeding study (EPA, 1984b). A 6-month dog feeding study established a NOEL of 60 mg/kg/day (EPA, 1984b). A systemic NOEL of 50 mg/kg/day was determined for a 107-week feeding study with rats (EPA, 1985d).

Teratology and multigeneration reproduction studies indicate that asulam does not cause teratogenic or fetotoxic effects in test animals. A 2-generation rat reproduction study also resulted in the absence of terata and fetotoxic effects at 1,000 ppm (50 mg/kg/day); however, reproductive effects characterized by a decrease in the number of live births were reported at 5,000 ppm (250 mg/kg/day) and 25,000 ppm (1,250 mg/kg/day) (EPA, 1984b). The highest NOEL reported for a rabbit teratology study was 300 mg/kg, with no teratogenic or fetotoxic effects reported (EPA, 1984b). A rat teratology study resulted in the absence of any toxic effects and a NOEL established at greater than 40 mg/kg/day (HDT) (EPA, 1984b).

Atrazine

Atrazine can be classified as a slightly toxic herbicide, based on the acute oral LD₅₀ of 1,869 mg/kg in rats, with labored breathing observed in surviving animals (EPA, 1984c). A 2-year dog feeding study resulted in the establishment of a systemic NOEL of 150 ppm (3.7 mg/kg/day) based on a decrease in body weight (EPA, 1984c). Atrazine has produced mammary tumors

in rats. A recently completed 2-year feeding/oncogenicity study in rats resulted in a systemic NOEL of 70 ppm (3.5 mg/kg/day) based on reduced body weight, reduced red blood cell parameters and decreased glucose levels (EPA, 1986). A 3-generation reproduction study in rats reported no reproductive or systemic effects at the highest dose tested (100 ppm or 5 mg/kg/day) (EPA, 1984c). Fetotoxic effects characterized as weight loss and fetal resorptions occurred at a dose level of 500 mg/kg in a rat teratology study (EPA, 1984c). A mouse teratology study reported no significant increases in fetal abnormalities at 46.4 mg/kg (Hayes, 1982).

Bromacil

Based on the acute oral LD₅₀ of 3,998 mg/kg in rats (EPA, 1984d), bromacil can be classified as slightly toxic. A systemic NOEL of 250 ppm (6.25 mg/kg/day) was derived from a 2-year dog feeding study that indicated decreases in body weights (EPA, 1984d). There have not been any teratogenic or fetotoxic effects reported from teratology studies that established a NOEL greater than 250 ppm (7.5 mg/kg/day) for rabbits and a NOEL greater than 7.92 mg/kg for rats (EPA, 1984d).

2,4-D

2,4-D can be classified as moderately toxic (see Table 3-1) in mammals with an LD₅₀ in rats of 375 mg/kg (EPA, 1984e). Acute and chronic toxicity studies in mammals revealed general systemic toxic effects following ingestion of large doses of 2,4-D. Similar clinical symptoms have been observed in human case reports. Even though dermal absorption of 2,4-D is limited, the herbicide has been reported to produce peripheral neuropathy in a few individuals after accidental exposure. In several cases the recovery has not been complete (USDA, 1984). A 2-year dog feeding study with dose levels of 2,4-D ranging from 0 to 500 ppm (0 to 12.5 mg/kg) established a systemic NOEL of 12.5 mg/kg/day (HDT) (EPA, 1984e). A systemic NOEL of 1,250 ppm (62.5 mg/kg/day) was established for a 2-year rat feeding study (EPA, 1984e).

Results from the first year of a chronic feeding study on rats have been reviewed by EPA (EPA, 1985c). Based on kidney effects, a NOEL of 1 mg/kg/day was established; the lowest effect level was 5 mg/kg/day. Based on this study using a hundredfold safety factor, EPA has established a provisional ADI of 0.01 mg/kg/day.

Fetotoxic and maternal toxic NOEL's of 5 mg/kg/day are based on a study with rats exposed to 2,4-D acid at 5, 20, and 80 mg/kg/day. Decreased maternal body weight and reduced pup weight were observed at 20 mg/kg/day (EPA, 1986b). There were no teratogenic effects observed in the offspring of rats exposed to 2,4-D (EPA, 1985c).

2,4-DP

2,4-DP can be classified as slightly toxic. Two studies reported LD₅₀'s of 650 mg/kg for mice and 532 mg/kg for rats (EPA, 1984f). A subchronic 90-day rat feeding study established a NOEL of 5 mg/kg. At higher doses, enzymes in the blood, hemoglobin, and kidney and liver weights were affected. A systemic NOEL of 100 mg/kg/day (EPA, 1984f) based on increased liver

weights was established for an 18-month mouse feeding study. Two-year feeding studies with rats determined a systemic NOEL of 50 mg/kg/day. At the LEL, all of the following effects were observed: decreased weight gain, decreased hematocrit and red blood cells, chronic prostatitis, and kidney degeneration (EPA, 1984f).

A fetotoxic NOEL of 125 ppm (6.25 mg/kg/day) was reported for a 3-generation rat reproduction study, with increased mortality of pups occurring at 500 ppm (25 mg/kg/day) (EPA, 1984f). A rabbit teratology study determined fetotoxic and maternal NOEL's of 25 mg/kg and a teratogenic NOEL of less than 25 mg/kg, which was the lowest dose tested. Teratogenic effects characterized by displaced kidneys, navel hernia, and distorted ribs occurred at 25 mg/kg in rabbits. Fetotoxic effects such as reduction in fetal weight and reduction in crown-rump distance were reported at a dose level of 100 mg/kg/day in rabbits. Maternal toxic effects such as unsteadiness in gait, reduced food intake, and mortality were also observed at the rabbit dose level of 100 mg/kg/day.

Dalapon

Based on the acute oral LD₅₀ of 7,577 mg/kg in the rat (EPA, 1984g), dalapon can be classified as slightly toxic (see Table 3-1). A systemic NOEL of 15 mg/kg/day was reported for a 2-year rat feeding study based on increases in kidney weights (Paynter et al., 1960, as cited in USDA, 1984). A systemic NOEL of 50 mg/kg/day was reported for a 52-week dog feeding study which also indicated an increase in kidney weights (Paynter et al., 1960, as cited in USDA, 1984). Histology sections of test animal tissues revealed no abnormal pathology or evidence of tumorigenesis due to exposure to dalapon (USDA, 1984). The lowest NOEL reported for teratology studies is 300 mg/kg/day for the rat (USDA, 1984). The only toxic effect noted to occur after maternal exposure to dalapon is reduced weight of pups. The Environmental Protection Agency has initiated a data call-in for a reassessment of the reregistration of dalapon (EPA, 1984g).

Dicamba

Based on an acute oral LD₅₀ of 757 mg/kg in the rat, dicamba can be classified as slightly toxic (USDA, 1984) (see Table 3-1). A number of subchronic rat studies and one 2-year rat study did not find any adverse effects at the highest dose tested (EPA, 1984h). EPA reports an in-house value of 250 mg/kg/day for systemic effects (EPA, 1985d). EPA has requested additional chronic studies for dicamba. A 90-day subchronic feeding study in rats established a NOEL of 500 ppm (25 mg/kg/day) based on slight liver cell alterations. This NOEL is similar to a NOEL from a chronic mouse study reported in DOE, BPA, 1984 and, because it is more conservative, will be used in this risk analysis. Fetotoxic and maternal toxic effects have been observed in laboratory animals exposed to dicamba. A fetotoxic NOEL of 0.5 mg/kg was reported for a rabbit teratology pilot study, with resorptions reported at 1.0 mg/kg (EPA, 1984h). A second rabbit teratology study resulted in a maternal NOEL and a fetotoxic NOEL of 3.0 mg/kg (EPA, 1984h). Recent information from EPA (1985d) has placed the reproductive NOEL of dicamba at 2.5 mg/kg.

Diuron

Based on the acute oral LD₅₀ of 3,750 mg/kg (EPA, 1984i), diuron can be classified as slightly toxic (see Table 3-1). A systemic NOEL of 125 ppm (6.25 mg/kg/day) was derived from a 2-year rat feeding study (EPA, 1984i). A systemic NOEL of 25 ppm (0.625 mg/kg/day) was reported for a 2-year dog feeding study (EPA, 1984i). Awaiting information from EPA on these studies. A 3-generation rat reproduction study resulted in the absence of any reproductive effects at the only dose tested of 125 ppm (6.25 mg/kg) (EPA, 1984i). In that same study, a systemic NOEL of less than 125 ppm (6.25 mg/kg/day) was established based on body weight depression of litters (EPA, 1984i).

Fosamine

Based on the acute oral LD₅₀ of 24,400 mg/kg in the rat for the formulated product (USDA, 1984), fosamine can be classified as very slightly toxic.

A systemic NOEL of 1,000 ppm (25 mg/kg/day) was reported for a 6-month dog feeding study, with increased stomach weight being the only toxic effect noted (Schneider and Kaplan, 1983, in USDA, 1984). A systemic NOEL between 5,000 and 10,000 ppm (250 mg/kg/day to 500 mg/kg/day) (HDT) was established for a 90-day rat feeding study (Schneider and Kaplan, 1983, in USDA, 1984).

NOEL's of 10,000 ppm (500 mg/kg/day) and 5,000 to 10,000 ppm (250 to 500 mg/kg/day) were reported for rat teratology studies (Schneider and Kaplan, 1983, in USDA, 1984). No fetal toxic, teratogenic, or reproductive toxic effects were observed in either of the teratology studies.

Glyphosate

Based on the acute oral LD₅₀ of 4,320 mg/kg (EPA, 1984k) in the rat, glyphosate can be classified as slightly toxic (see Table 3-1). A 26-month rat feeding study found no observable effects, including cancer, at the highest dose tested. Based on this study, EPA established a NOEL of greater than 31 mg/kg/day (HDT) (EPA, 1984k). A 3-generation reproduction study of glyphosate in rats established a NOEL of 10 mg/kg/day (EPA, 1984k). This NOEL was based on renal tubular dilation in the kidneys of the pups; no effects on fertility or reproductive parameters were noted. Based on this study, EPA has established an acceptable daily intake (ADI) level of 0.1 mg/kg/day. Maternal NOEL's for two teratology studies in rats and rabbits were 1,000 mg/kg/day and 175 mg/kg/day, respectively (EPA, 1984k).

Hexazinone

Hexazinone can be classified as slightly toxic based on the acute oral LD₅₀ of 1,690 mg/kg (EPA, 1984l). The systemic NOEL based on 2-year mice and rat feeding studies was established as 200 ppm (30 mg/kg/day, mice; 10 mg/kg/day, rats) (EPA, 1984l). The toxic effects observed during the mouse study were increases in liver size, a localized increase in liver cells, and localized tissue degeneration at the lowest effect level of 2,500 ppm (375 mg/kg/day). Twenty mg/kg/day was the lowest NOEL reported for rabbit

teratology studies (USDA, 1984). A 3-generation reproduction study established a NOEL of 1,000 ppm (50 mg/kg/day) in rats (USDA, 1984). A rabbit teratology study reported both a terata and fetotoxic NOEL greater than 125 mg/kg (HDT) (EPA, 1984l).

Picloram

Based on the acute oral LD₅₀ of 8,200 mg/kg in rats (EPA, 1984m), picloram can be classified as very slightly toxic. A 6-month dog feeding study, during which test animals were exposed to picloram at the dietary levels of 0, 7, 35, and 175 mg/kg/day, resulted in a chronic NOEL of 7 mg/kg/day (Barna-Lloyd et al., 1982, as cited in Mullison, 1985). Increased liver weights were reported at the lowest effect level of 35 mg/kg/day in males (DOW Chemical Company, 1984).

Two chronic feeding studies performed by IBT have been invalidated by EPA (1984m). EPA has asked for additional data on chronic rodent and nonrodent studies.

The lowest reproductive NOEL reported for picloram is 1,000 ppm (50 mg/kg/day) in rats, with reduced fertility at the lowest effect level of 3,000 ppm (150 mg/kg/day) (EPA, 1984m). In a rat teratology study, minor skeletal abnormalities related to maternal toxicity were observed at 750 mg/kg/day. No deformed offspring or adverse effects to development of the newborn offspring were observed at the highest dose tested (1,000 mg/kg). A teratology study using mice as the test species observed no adverse effects on fertility or number of offspring after exposure to 15 mg/kg/day of picloram (USDA, 1984).

Simazine

Based on the acute oral LD₅₀ of greater than 5,000 mg/kg in the rat (EPA, 1984n) simazine can be classified as very slightly toxic (see Table 3-1). A systemic NOEL greater than 100 ppm (5 mg/kg/day) (HDT) was reported for a 2-year rat feeding study (EPA, 1984n). A 2-year dog feeding study did not find any overt signs of toxicity at 1,500 ppm (37.5 mg/kg/day) (HDT). EPA, however, has determined that chronic toxicity and oncogenic potential could not be determined from either of these studies. A 3-generation rat reproduction study established a NOEL greater than 100 ppm (5 mg/kg/day); there were no reported teratogenic, fetotoxic, or reproductive effects at the highest dose tested (EPA, 1984n). EPA has requested additional studies on subchronic toxicity, teratogenicity, and chronic toxicity.

Tebuthiuron

Based on the acute oral LD₅₀ of 644 mg/kg in rats (Todd et al., 1974, in DOE, 1983), tebuthiuron can be classified as slightly toxic. A systemic NOEL of 554 ppm (83.1 mg/kg/day) (EPA, 1984o) was established from a 119-day mouse feeding study. A systemic NOEL of 500 ppm (12.5 mg/kg/day) was established for a 3-month dog feeding study based on increased thyroid-to-body weight values and increased blood enzyme levels. A reproductive NOEL of greater than 400 ppm (20 mg/kg/day) (HDT) was determined in a 2-generation reproduction study with rats (EPA, 1985d). Tebuthiuron did not produce any terata among the offspring of rats at the

highest dose tested of 1,800 ppm (90 mg/kg) (Todd et al., 1974, as cited in USDA, 1985b). A reproductive NOEL of 90 mg/kg/day is used in this risk assessment because neither of the studies reported above showed reproductive effects at the highest doses tested. A dermal teratology study in rats resulted in a NOEL of 237 mg/kg/day (EPA, 1984o).

Triclopyr

With an acute oral LD₅₀ ranging from 630 to 729 mg/kg in rats (EPA, 1984p), triclopyr can be classified as slightly toxic (see Table 3-1). A systemic NOEL of 30 mg/kg/day (HDT) was established for both rat and mouse 2-year feeding studies (EPA, 1984p; USDA, 1984). EPA does not consider this study adequate and has requested additional chronic studies. A 228-day dog feeding study resulted in a systemic NOEL of less than 5 mg/kg/day, based on decreased weight gain and food consumption (Dow Chemical Company, 1983, in USDA, 1984). A 6-month feeding study with dogs resulted in a systemic NOEL of 2.5 mg/kg (HDT) (40 CFR Part 180 5(84):18485 May 1, 1985). The effects found in the dog studies are not representative of effects expected in humans because dogs have a limited capacity for organic anion transport in the kidney. Dogs excrete triclopyr at a slower rate than other laboratory animals or humans. The half-life of triclopyr for urinary excretion in dogs is 96 hours as compared to 1.5 hours in rats and 3.1 hours in monkeys. Toxicity may be increased in dogs because of the greater relative retention time of the compound in the animal's body. Therefore, the use of the NOEL from the dog study (the lowest NOEL found in the literature) in this risk assessment is very conservative and tends to overestimate expected effects in humans.

Laboratory results indicate that triclopyr has caused mild fetotoxicity characterized by delayed ossification of the skull in the offspring of rats exposed to 200 mg/kg/day (USDA, 1984). No teratogenic or reproductive toxic effects were observed during a 3-generation rat reproduction study establishing a NOEL of greater than 30 mg/kg/day (HDT) (USDA, 1984). A NOEL less than 25 mg/kg/day was established for a rabbit teratology study in which there were no teratogenic effects reported, but fetotoxic effects were observed at the dose levels of 25, 50, and 100 mg/kg/day (USDA, 1984). A fetotoxic NOEL of less than or equal to 10 mg/kg was established in a study with rabbits (EPA, 1984p).

MUTAGENICITY OF THE 16 HERBICIDES

This subsection presents a review of the currently available information on the mutagenic hazard of the 16 herbicides. Table 3-3 summarizes the validated tests on each of the 16 herbicides for each category of testing recommended by EPA in their guidance documents on mutagenicity (EPA, 1978 and EPA, 1984r). Table 3-3 also presents the relevance of the recommended tests to a determination of human mutagenic potential according to Dr. David Brusick (see Section 6).

In general, the most relevant mutagenic assays are in vivo cell studies and germ cell or gonadal studies (e.g., Drosophila fruit fly sex-linked recessive studies). A germ cell study is considered relevant to evaluating

the mutagenicity of a chemical even if the test organism is not mammalian. In vitro studies using mammalian cells are of less importance because of the high percentage of false positive findings induced as a result of interactions between the cultured cells and media conditions. Tests for detecting primary DNA damage (Group 3 in Table 3-3) are not useful in determining the human mutagenic potential of a chemical.

For some of the herbicides, no validated mutagenicity tests exist or the mutagenicity tests conducted are insufficient to conclude whether the chemical is mutagenic. For these herbicides, the worst case analysis presented in Section 5 assumed that these herbicides are mutagenic. In these cases the results of carcinogenicity tests (see Table 3-4) were used to estimate mutagenic risk, because there is no widely accepted scientific method to quantify mutagenic risks directly from mutagenicity test data.

Details of the mutagenicity testing on the 16 herbicides are presented in Attachment A.

Amitrole

Amitrole did not produce mutagenic effects in 58 bacterial assays. Positive results were observed in two tests in an unvalidated system with unusual bacteria. EPA has determined that amitrole does not present a potential for genetic effects. The chemical induced transformations in four in vitro assays with mammalian cells (EPA, 1985a). EPA has stated that these results support cancer potential, but not necessarily mutagenic potential.

Asulam

There is no evidence to indicate that asulam has mutagenic potential. Asulam was nonmutagenic when tested in a bacterial assay and assays using mammalian cells in vitro and in vivo (EPA, 1984b).

Atrazine

Atrazine was positive for mutagenicity in eight gene mutation studies and negative in eight others. Three of these positive responses were in tests with the fruit fly that measured gene mutations in germ cells. Positive results were also obtained in tests with mice which measured chromosome alterations in germ cells. Positive responses in these types of assays indicate a potential for mutagenic hazard. Chromosome aberrations in bone marrow cells in vivo support this conclusion. However, these in vivo responses were observed only at very high levels of atrazine equal to or exceeding 1.5 gm/kg (USDA, 1984). Atrazine was nonmutagenic in two microbial mutagenicity studies accepted by EPA (1984c). Although these results show that atrazine must be viewed as mutagenic at high levels of exposure, the degree of hazard to humans from low levels of exposure would be minimal.

Bromacil

According to EPA (1982) in a letter to E.I. DuPont De Nemours Co., Inc. dated September 30, 1982, EPA has sufficient data to characterize the mutagenicity of bromacil. According to DuPont De Nemours Co., Inc. in their comments on the Draft of this document (DuPont, 1986), EPA considers bromacil to be nonmutagenic. DuPont states that in studies conducted by non-DuPont laboratories, bromacil has been reported to be nonmutagenic in 5 of 6 point/gene mutation studies, 6 of 6 primary DNA damage studies, and 3 of 4 tests for chromosomal effects.

Table 3-3
Mutagenicity Testing on the 16 Herbicides^a

Mutagenicity Test Type ^c		Herbicide																Value in Determining Human Mutagenicity ^b	
Group 1--Tests for detecting gene mutations																			
A.	Bacteria with and without metabolic activation	2(+)	56(-)	1(-)	3(+)	7(-)	2(+)	13(-)	4(-)	3(-)	1(-)	3(-)	2(-)	1(+)	5(-)	9(-)	1(-)		
B.	Eukaryotic microorganisms with and without metabolic activation	+			3(+)		1(+)	1(-)	1(+)			1(-)			3(-)				
C.	Insects (e.g., sex-linked recessive lethal test)	++	3(-)		1(+)	1(-)	1(+)	1(-)								2(+)			
D.	Mammalian somatic cells in culture with and without metabolic activation	++		1(-)	1(+)					1(-)	1(-)	1(-)	1(-)				1(+)		
E.	Mouse specific locus test in vivo	++																	
Group 2--Tests for detecting chromosomal aberrations																			
A.	Cytogenetic tests in mammals in vivo	++	2(-)		2(+)		1(+)	1(-)		1(-)		1(-)	1(-)						
B.	Insect tests for heritable chromosomal effects in vivo	++																	
C.	Dominant-lethal effects in rodents, heritable translocation tests in rodents, and in vitro cytogenetic assays in mammals	++	4(+)	1(-)	1(+)	3(-)	2(-)		1(-)	1(+)	2(-)	1(+)	1(+)				1(+)		
Group 3--Tests for detecting primary DNA damage																			
A.	DNA repair in bacteria (including differential killing of DNA repair defective strains) with and without metabolic activation	NA							2(+)	2(+)							1(-)		
B.	Unscheduled DNA repair synthesis in mammalian somatic cells in culture, with and without metabolic activation	NA			1(+)		2(-)		1(-)	1(-)	1(-)	1(-)	1(-)						
C.	Mitotic recombination and gene conversion in yeast, with and without metabolic activation	NA		3(+)	6(-)	2(+)	3(-)	1(+)	1(-)								2(-)		
D.	Sister-chromatid exchange in mammalian cells in culture, with and without metabolic activation	NA	2(-)		1(-)		1(+)										*		

^aNo validated mutagenicity studies are available for bromacil, dalapon, and diuron.

^bValue in Determining Human Mutagenicity Source: USDA, 1985a

^cSource: FIFRA, Environmental Protection Agency: Proposed Guidelines for registering pesticides in the U.S. Hazard Evaluation: humans and domestic animals. Fed. Reg. 43:37335-37403, August 22, 1978.

NA = Not Applicable

+ = Applicable

++ = Greater applicability

* = Inconclusive

Source for Mutagenicity Data: USDA, 1984; EPA, 1985d

2,4-D

Mutagenicity studies on 2,4-D have indicated negative, weakly positive, and positive results for various test systems. Newton and Dost (1981) concluded that 2,4-D may be a weak mutagen but that it is "without significance as an environmental mutagenic hazard." EPA has requested additional data to evaluate the mutagenic potential of 2,4-D in mammalian test systems. The worst case assumption is that 2,4-D is mutagenic.

2,4-DP

A limited assessment of the genotoxicity of 2,4-DP is available. 2,4-DP was nonmutagenic when tested in three nonactivated microbial assays (EPA, 1984f). 2,4-DP was not mutagenic in the Ames test. A bacterial assay was positive for induction of repairable DNA damage with metabolic activation. Positive results were obtained when 2,4-DP was tested in two studies with yeast and a bacterial assay for unscheduled DNA synthesis (EPA, 1984f).

Based on the above data, it cannot be presumed that there is mutagenic hazard because in vitro or submammalian assays are inappropriate for hazard assessment. Based upon the inconsistent genotoxic responses and the positive oncogenic effects observed in a chronic oncogenic feeding study of rats, the worst case assumption is that 2,4-DP is mutagenic.

Dalapon

There are no validated mutagenic data on dalapon. The Environmental Protection Agency has initiated a data call-in including mutagenicity testing for a reassessment of the reregistration of dalapon (EPA, 1984g). Dalapon is probably not a mutagen because a 2-year rat feeding study resulted in no oncogenic effects (USDA, 1984). However, the worst case assumption is that dalapon has the potential to cause mutagenic effects.

Dicamba

Dicamba was nonmutagenic when tested in various microbial assay systems and a dominant lethal assay with mice (USDA, 1984). It is concluded that dicamba is nonmutagenic.

Diuron

There are no validated mutagenic studies on diuron. EPA has requested additional studies in this area. Du Pont has submitted four mutagenicity studies to EPA for the reregistration of diuron. According to Du Pont, diuron was nonmutagenic when exposed to a microbial assay, an in vitro mammalian cell assay, and the unscheduled DNA synthesis assay. Diuron induced chromosome aberrations in an in vivo mammalian assay (Du Pont, 1986). Diuron was shown to be nononcogenic in a long-term study; therefore it is not likely to be mutagenic. However, the worst case assumption is that diuron has the potential to produce mutagenic effects.

Fosamine

Fosamine was nonmutagenic when tested in bacterial assay systems, a cytogenetic assay using mammalian cells in vivo, and a DNA assay using mammalian cells in vitro (Schneider and Kaplan, 1983, as cited in USDA, 1984). Chromosome damage was induced by fosamine in an in vitro chromosome assay with rodent cells (Schneider and Kaplan, 1983, in USDA, 1984). Overall, fosamine is considered nonmutagenic and presents no mutagenic hazard to humans.

Glyphosate

Glyphosate was nonmutagenic in microbial assays and mammalian cell assay systems both in vitro and in vivo (EPA, 1984k). There is no evidence to indicate that it is mutagenic or presents any mutagenic risk to humans.

Hexazinone

Negative results were obtained from five of six mutagenicity test systems. Hexazinone was nonmutagenic in Ames assays, an in vitro mammalian cell assay, an assay of unscheduled DNA repair synthesis in mammalian somatic cells, and a mammalian cytogenetic assay (EPA, 1984l; USDA, 1984). Hexazinone was positive for inducing chromosome damage in an in vitro cytogenetic assay with rodent cells (USDA, 1984). This positive effect was observed only at very high levels and could be caused by a secondary effect, such as high ionic concentrations or pH. Based on these results, hexazinone is determined not to present mutagenic hazard to humans.

Picloram

Picloram was nonmutagenic in microbial assay systems and in the rat in vivo cytogenetic assay (USDA, 1984, and EPA, 1984m). Picloram was mutagenic in one assay on a previously untried system (USDA, 1984). The test used has not been validated for use in the standard battery of tests for mutagenicity. EPA has determined that the positive study was insensitive and not capable of determining mutagenicity in the test system. There is no evidence to conclude that it presents a mutagenic risk to humans. EPA has requested additional picloram mutagenicity studies. The worst case assumption is that it is a mutagen.

Simazine

None of the available mutagenicity studies have been validated by EPA (EPA, 1985d). Simazine was negative for mutagenicity when tested in microbial assay systems and in an in vitro mammalian DNA assay (USDA, 1984). A weakly mutagenic response and an increase in dominant lethals resulted from two studies with the fruit fly (USDA, 1984). This indicates that simazine may be mutagenic in some test systems and that it is a potential mutagenic hazard to humans.

Tebuthiuron

Tebuthiuron tested negative in one mutagenicity assay with bacteria and was weakly positive in a mammalian somatic cell test (EPA, 1985d). Tebuthiuron was nononcogenic in long-term laboratory studies and therefore is probably not a human mutagen. However, the worst case assumption is that tebuthiuron does have potential to cause mutagenic effects.

Triclopyr

Triclopyr was nonmutagenic in microbial assay systems and in a dominant lethal study using mice. A dominant lethal assay with rats was weakly mutagenic (USDA, 1984). Triclopyr may be mutagenic in some test systems, and is considered a potential human mutagen in this risk assessment.

CARCINOGENICITY OF THE 16 HERBICIDES

The following discussion summarizes the results of cancer tests and other chronic tests that have been used to determine whether any of the 16 herbicides are carcinogenic. Table 3-4 presents a summary listing of those results derived from chronic studies.

For those herbicides that have had extensive cancer testing or for which there is scientific controversy concerning the results of one or more cancer studies, the details of the cancer testing are presented in Attachment A. The next subsection on cancer potency summarizes the results of the analysis of tumor data on the five herbicides that have tested positive in at least one cancer study: amitrole, atrazine, 2,4-D, 2,4-DP, glyphosate, and picloram.

Amitrole Carcinogenicity

The many lifetime laboratory animal feeding studies on amitrole have consistently demonstrated its oncogenic potential. In feeding studies using rats, the thyroid and pituitary were the primary target organs at doses as low as 1/20 ppm amitrole (Food Drug Research Lab, 1981, as cited in EPA, 1985a). In mice, amitrole's oncogenic potential has not been as clearly demonstrated because liver and thyroid tumors occurred only when doses exceeded 2,000 ppm (Innes et al., 1969).

In a 2-year study comparing three animal species, doses of 100 ppm amitrole in the diet produced an increased incidence of thyroid tumors in rats but not in mice or hamsters (Steinhoff, 1983, as cited in EPA, 1985a). In addition, positive results in mice and hamster in vitro cell transformation studies have demonstrated amitrole's carcinogenic potential. Based on the available evidence, EPA has classified amitrole as a "probable human carcinogen." The epidemiological studies on humans do not qualify as "at least limited evidence of carcinogenicity to humans" because the authors' follow-up evaluation is entirely inconclusive. Thus, a higher classification is not warranted (EPA, 1985a). An analysis of its cancer potency is given in the next subsection.

Table 3-4

Summary of Mutagenicity and Carcinogenicity of Pesticides

Herbicide	Mutagenicity	Oncogenic Results from Chronic Studies
Amitrole	Nonmutagenic 63/69 assays (USDA, 1984) Amitrole does not present potential for heritable genetic effects (EPA, 1985a)	A probable human carcinogen (EPA, 1985a)
Asulam	Nonmutagenic in 1 assay (EPA, 1984b)	Oncogenic in 1 study (EPA, 1985d). Nononcogenic at HDT in 2 studies (EPA, 1984b)
Atrazine	Mutagenic in 19/38 assays (USDA, 1984)	Oncogenic in 1/3 studies (EPA, 1984c; EPA 1986)
Bromacil	Not considered mutagenic by EPA. Existing studies adequate (EPA, 1982). Nonmutagenic in 14/16 assays (DuPont, 1986)	Oncogenic in 1/2 studies (EPA, 1984d; EPA, 1985e)
2,4-D	Nonmutagenic in 28/43 assays (USDA, 1984)	Nononcogenic in 3 studies (EPA, 1984e; Hazelton Laboratories, 1986); Scientific uncertainty (Rueber, 1979, as cited in BLM, 1985); Oncogenic in 1 study, preliminary findings (EPA, 1986c)
2,4-DP	Nonmutagenic in 4/8 assays (EPA, 1984f)	Oncogenic in 1/2 studies (EPA, 1984f)
Dalapon	No studies reported (EPA, 1984g; USDA, 1984)	Nononcogenic in 2 studies (USDA, 1984);
Dicamba	Nonmutagenic in 6/8 assays (USDA, 1984)	Nononcogenic in 2 studies (EPA, 1984h); Studies not adequate according to EPA (EPA, 1985d). Nononcogenic in 1 study (EPA, 1986a)

Table 3-4 (Cont.)

Herbicide	Mutagenicity	Oncogenic Results from Chronic Studies
Diuron	No studies reported (EPA 1984i; USDA, 1984)	Nononcogenic in 2 studies (EPA, 1984i); Studies not adequate according to EPA (EPA, 1985d).
Fosamine	Nonmutagenic in 4/5 assays (USDA, 1984)	No chronic studies available. (EPA, 1984j; USDA, 1984)
Glyphosate	Nonmutagenic in 7 assays (EPA, 1984k)	Nononcogenic in 1 study at HDT (EPA, 1984k); possibility of weak oncogenic effect in mouse study (EPA, 1985d); scientific uncertainty (EPA, 1986c)
Hexazinone	Nonmutagenic in 4/5 test systems (USDA, 1984)	Nononcogenic in 2 studies (EPA, 1984l)
Picloram	Nonmutagenic in 9/10 assays (USDA, 1984)	Oncogenic in 1/2 studies (EPA, 1984m)
Simazine	Nonmutagenic in 12/14 studies, (USDA, 1984)	Nononcogenic in 1 study (EPA, 1984n)
Tebuthiuron	Nonmutagenic in 1/2 studies, (EPA, 1985d)	Nononcogenic in 1 study (USDA, 1984b)
Triclopyr	Nonmutagenic in 4/5 bacterial and cytogenetic assays (USDA, 1984; EPA, 1985d)	Nononcogenic in 3 studies (USDA, 1984)

Asulam Carcinogenicity

Three oncogenic feeding studies were validated by EPA (1984b, 1985d). An 18-month feeding study, during which mice were fed doses of asulam at levels of 1,500 and 5,000 ppm, resulted in nononcogenic effects being observed at either of the doses tested. A NOEL of less than 1,500 ppm (225 mg/kg/day) was reported for systemic toxic effects, such as decreased thyroid weights and increased kidney and heart weights, which were observed at both dosage levels. Another 18-month mouse oncogenic study also reported on the absence of oncogenic effects of 5,000 ppm (750 mg/kg/day), the highest dose tested.

A statistically significant increase in thyroid cell carcinomas and parafollicular cell carcinomas were observed in a rat study at 1,000 ppm (50 mg/kg/day) and 25,000 ppm (1,250 mg/kg/day) (EPA, 1983; EPA, 1985d). The rats received doses of 0, 50, 250, and 1,250 mg/kg/day during the 197-week feeding study. All effects reported were observed with female rats. An increase in adrenal medullary hyperplasia at doses of 250 and 1,250 mg/kg/day also was observed.

Atrazine Carcinogenicity

Available data suggest that atrazine may be carcinogenic. In an 18-month mouse feeding study, atrazine did not induce any tumors at 12.5 mg/kg/day (Innes et al., 1969).

A 2-year oncogenic feeding study of rats resulted in a significant increase in mammary tumors upon exposure to the dosage levels of 70, 500, and 1,000 ppm (3.5, 25, and 50 mg/kg/day). Based on the occurrence of mammary tumors in female rats, atrazine appears to be carcinogenic in rats (USDA, 1986a). Atrazine's cancer potency is discussed in the next section.

A formulated mixture of atrazine and simazine (Fogard-S) injected into mice produced malignant tumors of the lymph glands (Donna et al., 1981). However, because there were many flaws in this study and it is not possible to distinguish what caused the tumors, this study is not considered substantive evidence for carcinogenicity in atrazine. In addition, there is no reason to conclude that a carcinogenic nitrogen derivative of atrazine, N-Nitrosoatrazine (NNA), would be formed in toxic quantities in exposure of humans to atrazine. Details concerning Fogard-S and NNA are given in Attachment C.

Bromacil Carcinogenicity

Bromacil was carcinogenic in one of two oncogenicity studies conducted. No oncogenic effects were observed in rats as a result of exposure to bromacil for a 2-year test period (EPA, 1984d). EPA considers bromacil a liver carcinogen in CD-1 mice. Liver tumors in mice were observed at 5,000 ppm (750 mg/kg/day) after 18 months of exposure (EPA, 1985e).

2,4-D Carcinogenicity

A number of studies have assessed the carcinogenicity of 2,4-D, and thus far, there are no conclusive data demonstrating the carcinogenicity of 2,4-D (International Agency for Research on Cancer, 1977; Mullison, 1981; State of Minnesota, 1978, all as cited in USDA, 1984). However, there is also general agreement that none of these studies were adequate (EPA, 1982a; International Agency for Research on Cancer, 1977, as cited in USDA, 1984; WHO, 1984). At least one scientist, Dr. M. Rueber, disputes the conclusion that a carcinogenic effect of 2,4-D has not been shown. A preliminary review of the most recent cancer study by EPA indicates positive evidence of cancer in rats (EPA, 1986c). At 106 weeks, a preliminary report from a recent mouse study found that 2,4-D was not oncogenic at dosages of 1, 15, and 45 mg/kg/day (Hazelton Laboratories, 1986). An epidemiology study found no association between exposure and soft-tissue sarcoma or Hodgkin's disease in men exposed to agricultural herbicides, but observed a significant association for non-Hodgkin's lymphoma and phenoxyacetic acid herbicide exposure, especially 2,4-D acid exposure (Hoar et al., 1986). Details of these studies are given in Attachment A. In view of the uncertainties of 2,4-D carcinogenicity, 2,4-D was assumed to be carcinogenic in this risk assessment.

2,4-DP Carcinogenicity

Available evidence indicates that 2,4-DP is carcinogenic in rats (EPA, 1982b). A 2-year feeding study with rats showed tumor formation occurred at doses as low as 25 mg/kg/day (EPA, 1984f). At all doses tested (25, 50, or 150 mg/kg), malignant tumors were induced in test animals. Another study using mice as the test species showed no oncogenic effects at the highest dose tested (300 mg/kg/day) (EPA, 1984f). 2,4-DP is assumed to be a human carcinogen for the purposes of this analysis, and a risk assessment is presented in Section 5. 2,4-DP's cancer potency is discussed in the next subsection.

Dalapon Carcinogenicity

Available data do not indicate that dalapon is carcinogenic. In a 2-year rat feeding study (Paynter et al. 1960 as cited in USDA, 1984) and a 52-week dog feeding study (Paynter et al. 1960 as cited in USDA, 1984), histology sections of test animal tissues revealed no abnormal pathology or evidence of tumor formation resulting from exposure to dalapon (USDA, 1984).

Dicamba Carcinogenicity

Available evidence does not indicate that dicamba is carcinogenic. A 2-year rat feeding study resulted in the absence of any toxic or oncogenic effects of dicamba at 500 ppm (25 mg/kg/day) (HDT). This study is graded as supplementary data by EPA (EPA, 1984h). No oncogenic effects were reported in a 2-year dog feeding study (EPA, 1984h). EPA has requested additional cancer studies for dicamba because the available studies are not considered adequate for defining the oncogenic potential of dicamba based on EPA guidelines under FIFRA (EPA, 1985d). A recent 2-year rat study accepted by EPA (1986a) showed no oncogenic or systemic effects at the highest dose tested (2,500 ppm).

Diuron Carcinogenicity

Available evidence does not indicate that diuron is carcinogenic. At levels as high as 2,500 ppm, there was an absence of oncogenic effects in test animals exposed to diuron (Hodge et al., 1967; DOE, 1983). EPA has requested additional cancer studies on diuron to adequately determine the cancer potential of diuron under the current FIFRA guidelines.

Fosamine Carcinogenicity

Available evidence does not indicate that fosamine is carcinogenic. In a 6-month dog feeding study no oncogenic effects were observed (Schneider and Kaplan, 1983, in USDA, 1984). A 90-day rat feeding study showed no oncogenic effects (Schneider and Kaplan, 1983, in USDA, 1984). Neither the dosage levels administered nor the dosage levels at which toxic effects occurred were reported in 2-year feeding studies to evaluate the oncogenic potential of fosamine.

Glyphosate Carcinogenicity

A 26-month rat feeding study found no oncogenic effects at doses up to 31 mg/kg day (EPA, 1984k). However, this study has been downgraded to supplementary by EPA because the maximum tolerated dose (MTD) was not reached at the high dose. Kidney tumors were found at a high dose level in male mice in a glyphosate cancer study. However, the EPA Science Advisory Panel has reviewed all relevant data and concluded there is not sufficient evidence to conclude that glyphosate is oncogenic in the mouse (EPA, 1986b).

Although EPA has not concluded that glyphosate is a carcinogen based on the available data, for the purposes of this risk analysis, a worst case assumption is made that glyphosate is a carcinogen, and a risk assessment was conducted. EPA's position on glyphosate's carcinogenicity is presented in Attachment A. Glyphosate's cancer potency is discussed in the next subsection and in Attachment A.

A carcinogenic nitrogen derivative of glyphosate, N-Nitrosoglyphosate (NNG), is not considered a potential human hazard here because NNG is not likely to form in soils at the application rates used in forestry. Details concerning NNG are presented in Attachment A.

Hexazinone Carcinogenicity

Available evidence does not indicate that hexazinone is carcinogenic. In 2-year mouse and rat feeding studies no oncogenic effects of hexazinone were observed at any of the doses tested (200, 1,000, and 2,500 ppm in rats, and at the testing levels of 200, 2,500, and 10,000 ppm in mice) (EPA, 1984l).

Picloram Carcinogenicity

There has been disagreement among experts on the interpretation of studies about the potential of picloram to cause cancer. A rat oncogenicity study, in which test animals were exposed to an average of 14,875 ppm (743 mg/kg/day), was negative for oncogenic effects in males. However, liver tumors were observed in females (EPA, 1984q). A mouse oncogenicity study showed no tumor formation at dietary exposure levels ranging from 5,000 to 15,000 ppm (750 mg/kg to 2,250 mg/kg) (EPA, 1984q). Because of the positive female rat results, a cancer risk analysis has been conducted on picloram in this risk assessment under the assumption that picloram is carcinogenic.

Simazine Carcinogenicity

A chronic study conducted on simazine did not indicate that simazine is carcinogenic (Donna et al., 1981). EPA has requested additional data to assess the possible carcinogenicity of simazine.

The study on the atrazine-simazine mixture (Fogard-S) described above is not considered substantive evidence of the carcinogenicity of either of the constituents. Attachment A presents the details of that study.

Tebuthiuron Carcinogenicity

Available evidence does not indicate that tebuthiuron is carcinogenic. No carcinogenic effects were observed in a chronic feeding study in which rats and mice were fed from 0 to 1,600 ppm tebuthiuron in their diets for 2 years (Elanco Products Co. as cited in USDA, 1984b).

Triclopyr Carcinogenicity

Available data do not indicate that triclopyr is carcinogenic. For both rat and mouse 2-year feeding studies (Dow Chemical Company, 1983b, in USDA, 1984) and a 228-day dog feeding study no oncogenic effects occurred in test animals exposed to triclopyr.

CANCER POTENCY

This subsection presents the results of the cancer potency analysis for each of the herbicides assumed to be carcinogenic in this risk assessment. (See Attachment A for details of this analysis.) The cancer potency value is used later in the risk analysis to determine the human cancer risk under specified assumptions about lifetime human exposure.

The cancer potency of a chemical is defined as the increase in likelihood of getting cancer from a unit increase in the dose of the chemical. This relationship is illustrated by the graph of Figure 3-2. The line specifies what the increase in cancer probability is for each unit increase in dose in mg/kg/day.

The cancer potency is derived from tumor data generated in laboratory animal studies. Note in Figure 3-2 that the dose levels used in the laboratory cancer studies are high but those liable to be experienced by humans through exposure in the environment are low. Note also that the line relating dose to cancer probability approximates a straight line in the low dose region.

The following paragraphs give the specific sources of data used for the cancer potency estimates. Some of the potency estimates were made using the Global 82 computer program as given by EPA or by Crump (1983). The others were calculated from the referenced data using a least-squares linear regression procedure. The one-hit model was expressed as follows for the regression:

$$- \ln (1 - P(d)) = a + (b \times d)$$

where d is the average daily dose over a lifetime in mg/kg/day and $P(d)$ is the probability of cancer as a result of dose d .

The slope estimated by the regression, b , is an estimate of the cancer potency for the test animal. It should be noted that the least-squares estimates of a and b are also maximum-likelihood estimates under this model. In all cases we used a 95-percent upper confidence limit for ' b '. The parameter ' a ' represents the background level of tumors in the test animal, so it did not enter into the calculations of cancer risk for humans.

Several aspects of this analysis make the cancer potency estimates very high (pessimistic). First, it is assumed that any dose, no matter how small, has some probability of causing cancer. This is the nonthreshold property, discussed previously, indicating that even a single, extremely small dose may be enough to trigger cancer. Amitrole, for example, has been shown to cause cancer in test animals only at relatively high doses. EPA recommends using a threshold approach to analyze amitrole's carcinogenicity, but this analysis uses the nonthreshold assumption for all seven herbicides.

Second, one of the principal areas of scientific controversy in cancer risk assessment is in extrapolating from the high doses used in animal studies to the far lower doses humans may get. Models other than the one-hit model, which assumes a straight line at low doses as illustrated in Figure 3-2, have been used for the extrapolation of cancer data to assess human risk. However, the one-hit model used in this analysis gives the highest estimate of cancer potency and, in turn, cancer risk at the low doses liable to be seen in exposed humans.

Third, the cancer potency used in the calculation of human risk in this analysis is not the expected potency value, but the upper limit value of the 95-percent statistical confidence interval. Using this upper limit value gives a potency that is approximately twice as high as the expected potency estimate. The details of the cancer potency derivation for all of the chemicals based on the one-hit model are given in Attachment A.

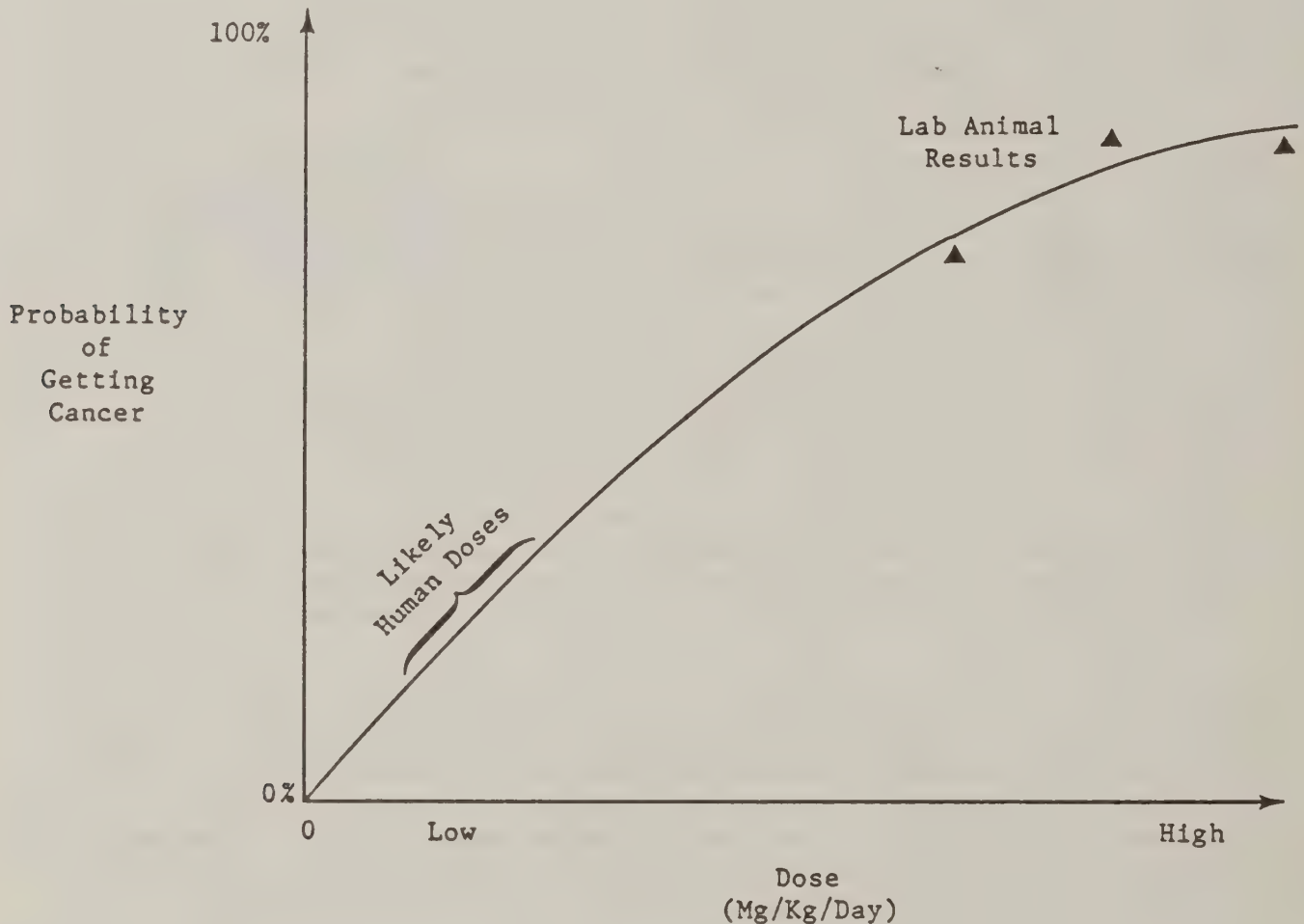


Figure 3-2 Cancer Potency Curve

Amitrole Cancer Potency

Amitrole cancer potency was estimated using data from three studies:

1. A 2-year rat feeding study conducted by Hazelton Laboratories, Inc.
2. A study by Tsuda et al. (1976) in which rats were given 2,500 ppm in their drinking water.
3. A study by Food and Drug Research (1981, as cited in EPA, 1985a) in which rats alternately were fed food with and without amitrole.

The cancer potency for amitrole estimated from the Hazleton Labs rat study data was 0.15 per (mg/kg/day). The data of Tsuda et al. (1976) gave a potency of 0.011 per (mg/kg/day) for all invasive thyroid lesions and 0.00098 per (mg/kg/day) for papillary adenoma. The Food and Drug Research 1981 study (as cited in EPA 1985a) indicated a cancer potency for thyroid tumors of 0.61 (considering only the intermittently dosed groups). In this risk assessment, the highest of these potencies is used to estimate human cancer risk. The 95-percent upper confidence limit for the potency based on the Food and Drug Research data is 1.4 per (mg/kg/day).

Asulam Cancer Potency

Asulam cancer potency was based on the rate of tumor formation in thyroid cells in male rats in a 107-week feeding study (EPA, 1983). The cancer potency using the one-hit model is 0.02 per (mg/kg/day).

Atrazine Cancer Potency

Atrazine cancer potency was calculated based on the rate of mammary tumor formation in female rats in a 2-year chronic feeding oncogenicity study (EPA, 1986). The cancer potency estimated using the single hit cancer model is .03 per (mg/kg/day) (USDA, 1986).

Bromacil Cancer Potency

Bromacil cancer potency was based on the rate of liver tumor formation in male mice in an 18-month feeding study. The cancer potency estimated from the one-hit model is 0.0038 per (mg/kg/day) (EPA, 1985e).

2,4-D Cancer Potency

2,4-D cancer potency was calculated based on the rate of tumor formation in the female Osborne-Mendel rats studied by Hansen et al. (1971). This is the species and sex that have exhibited the highest increase in tumor formation after 2,4-D administration. All tumors were considered, although many of them were benign. As in the case of amitrole, the conservative one-hit model was used to represent the relationship between dose and rate of tumor formation. The 95-percent upper confidence limit of the cancer potency, calculated by Crump (1983) using the GLOBAL 82 computer program, was 0.00503 per (mg/kg/day). EPA has studied that a preliminary review of an additional long-term oncogenicity study submitted to EPA in 1986 indicates that the cancer potency level would be of about the same magnitude as the cancer potency calculated by Crump (EPA, 1986).

2,4-DP Cancer Potency

A cancer study involving rats fed up to 200 mg/kg (EPA, 1982b) was used to derive 2,4-DP cancer potency. In this study, the highest dose group showed signs of general toxicity because they were fed more than the maximum tolerated dose of 2,4-DP. Many of the females at all dose levels had tumors but they did not show a dose-related response. Males showed a significant increase in the rate of incidence of malignant tumors, with a corresponding decrease in the rate of benign tumors. The tumors were primarily in the thyroid and pituitary glands.

The 95-percent upper confidence limit for the cancer potency of 2,4-DP was estimated from the male rat data as 0.059 per (mg/kg/day). Only malignant tumors were considered in this case, and the high dose group showing signs of general toxicity was not considered in order to give the highest cancer potency indicated by the data. The high dose group actually had fewer malignant tumors than the intermediate dose group.

Glyphosate Cancer Potency

Glyphosate cancer potency was based on the rate of kidney tumor formation in male mice in the feeding study reported in EPA (1985b).

The upper 95-percent limit of the cancer potency of glyphosate calculated from the kidney tumor data was 0.000034 per (mg/kg/day).

Picloram Cancer Potency

The Gulf Research Institute conducted a carcinogenic bioassay of picloram in rats and mice for the National Cancer Institute (1978). There was evidence that picloram affected the livers of male and female rats, and the study concluded that the findings were "suggestive of ability of the compound to induce benign tumors in livers of female Osborne-Mendel rats."

Using the one-hit model, a 95-percent upper confidence limit on picloram carcinogenicity has been calculated by Crump (1983) using the GLOBAL 82 computer program. His estimate is 0.00057 per (mg/kg/day). This is only about one-tenth as great as that for 2,4-D.

INERT INGREDIENTS

Inert ingredients are chemicals used with the active ingredient in preparing a formulation of a herbicide. Inert ingredients are used to provide a carrier for the active ingredient that facilitates the effective application of the herbicide. Inerts are not intended to supplement the herbicide's toxic properties.

EPA's Office of Pesticide Programs (EPA 1986d) has identified about 1,200 inert ingredients that are now used in approved pesticides and has reviewed the existing evidence concerning the toxicity of these inerts, including laboratory toxicity data, epidemiological data, and structure/activity relationships. Of particular concern in reviewing the inerts was their potential for causing chronic human health effects. The EPA review resulted in categorizing the 1,200 inerts into four lists.

List 1 contains about 55 inerts that have been shown to be carcinogens, developmental toxicants, neurotoxins, or potential ecological hazards and that merit the highest priority for regulatory action.

List 2 contains approximately 50 chemicals that have been given high priority for testing because toxicity data is suggestive, but not conclusive, of possible chronic health effects or because they have structures similar to chemicals on List 1.

List 3 contains about 800 chemicals that are of lower priority because no evidence from toxicity data or from a review of their chemical structure would now support a concern for toxicity or risk.

List 4 of about 300 chemicals contains those inerts generally recognized as safe.

Because EPA normally classifies inert ingredients as "Confidential Business Information", information on them does not have to be released by EPA to the public under the Freedom of Information Act (See also 40 CFR 1506.(a)). Nonetheless, the BLM requested that the EPA review the herbicides proposed for use, and disclose whether any of them contained inert ingredients of or suggesting toxicological concern. EPA has completed this review.

The EPA has identified only one inert ingredient on either list 1 or list 2. This ingredient, contained in Esteron 99, is a petroleum distillate of high priority for testing. Accordingly, a risk analysis has been conducted on the human health risk from exposure to the petroleum distillate in Esteron 99. The risk analysis also considers risk from exposure to diesel oil, a petroleum distillate used as a carrier for herbicides. Other than petroleum distillates, the BLM intends to use only formulations containing inerts that EPA generally recognizes as safe or that do not support a specific concern for toxicity or risk.

EPA (1987) noted that concerns regarding the acute toxicity of inert ingredients are usually addressed through tests of the herbicides as formulated products. While the herbicides as formulated products have undergone acute toxicity testing, they generally have not undergone extensive chronic toxicity testing, or cancer, reproductive, developmental or mutagenicity tests. The gap in the testing of the herbicides as formulated products, according to one view, gives rise to an inference that the environmental consequences, including hazards to human health, from using them are largely unknown. This theory holds that regardless of what is known about each herbicide formulation's two components, that is, the active ingredients and inerts, the possibility exists that the formulated product may pose greater risk than separate consideration of each component may suggest. Given the little information that is available on each herbicide's formulation, the possibility can not be discounted entirely. Neither can it be presumed as true. The possibility that the herbicides' formulations may pose greater risk than their components is largely an untested hypothesis. This possibility has not been shown to be true, however, regarding the herbicides' formulations acute toxicity. Turning to the competing viewpoint, and the one adopted in this analysis, the data gaps about the herbicides as formulated products are considered insignificant since the risks posed by the herbicides active ingredient are over stated. Any risk posed by the herbicides as formulated products is considered to be embraced in the analysis of the active ingredients. Moreover, each herbicide as a formulated product contains two types of ingredients: active and inert. Each type of ingredient has known and suspected properties. The herbicides' active ingredients have undergone cancer, reproductive, developmental and mutagenicity tests of varying degrees. The herbicides' inerts have undergone categorization according to their toxicity and risks, if any. With only one lone exception, no specific concern exists with the herbicides' inerts. Thus, because the herbicides' active ingredients here, not their inerts, pose the risks, it logically follows that any analysis drawing attention to the former as opposed to the latter is properly focused.

An analysis of the hazard of Esteron 99's inert ingredient of toxicological concern and diesel oil now follows.

Esteron 99 contains petroleum distillates as inert ingredients. The toxicity of the petroleum distillates can be estimated from the toxicity of diesel.

Diesel oil is a complex variable mixture of hydrocarbons with a boiling point range of from 350° to 700° F and an aromatic content ranging up to 35 percent (DOE 1983). Diesel fuel is usually a straight-run distillation product that boils below 650° F, contains few polycyclic aromatics, and has not been shown to be carcinogenic. A 2-year oncogenic skin-painting study (terminated after 62 weeks), during which Swiss Epley mice were exposed to 0.05 mL (41 mg) of diesel fuel products, resulted in skin carcinomas in 2 of 50 animals. These results were not statistically significant by chi-square analysis. The study was prematurely terminated because of the presence of extensive skin lesions in test animals (American Petroleum Institute 1983). Higher boiling point (greater than 700° F) petroleum products subjected to more refinement processes, such as cracking or hydrogenation, and that contain polycyclic aromatics may be carcinogenic to experimental animals (Bingham and others 1979).

Beck and others (1982) conducted a short-term exposure study examining the acute toxicity of 19 petroleum hydrocarbons in acute oral, acute dermal, subacute dermal, and eye irritation studies. On the basis of an acute oral LD₅₀ of 9.0 mL/kg (7,380 mg/kg), diesel oil can be classified as a very slightly toxic compound. The LD₅₀ is about 20 times greater than that of 2,4-D. The most marked acute toxic effect observed after the administration of oil to test animals occurred during diesel primary dermal irritation studies. A single diesel oil exposure to rabbits resulted in a rating of "extremely irritating" based on a score of 6.82 (on a scale of 1 to 10). Irritation may have been caused by chemical additives used to make diesel oil burn more efficiently in internal combustion engines. Diesel oil was nonirritating in primary eye irritation studies. A subacute 3-week dermal study of eight rabbits resulted in an average weight loss of 0.38 kg at a dose level of 4.0 mL/kg (3,280 mg/kg) and an average weight loss of 0.55 kg with a 67-percent mortality rate at a dose level of 8.0 mL/kg (6,560 mg/kg).

An inhalation teratology study in which rats were exposed to 101.8 ppm or 401.5 ppm (5.09 or 20.075 mL/kg) of diesel fuel on days 6 through 15 of gestation resulted in no significant teratogenic effects (Mecler and Beliles 1979 as cited in American Petroleum Institute 1983). Diesel fuel was nonmutagenic when tested in the Ames assay and the mouse lymphoma assay, but it was found to be clastogenic (causing chromosomal breaks) in rat bone marrow cells (Conaway and others 1982). Because diesel oil contains polycyclic aromatic hydrocarbons and other constituents that are known or suspected mutagens, this risk assessment considered it a mutagen as a worst-case assumption.

Diesel oil has not been shown to be carcinogenic, but it is a complex mixture that typically contains small amounts of substances known or suspected of being carcinogenic. For the purposes of this risk assessment, diesel oil is assumed to be carcinogenic because of its benzene content and its content of polycyclic aromatic hydrocarbons, typified by the potent carcinogen benzo(a)pyrene. But these carcinogens occur in such small amounts that they do not contribute significantly to the potency calculated for 2,4-D. Consequently, cancer risk was not calculated separately for petroleum oil but only for the total mixture with 2,4-D.

The oncogenic potential of petroleum fuels is directly related to refinery processing methods used to obtain the petroleum product and the crude oil composition from which the fuel was derived. An evaluation of the composition of petroleum fuels has revealed a positive correlation between polycyclic aromatic hydrocarbon (PAH) content and carcinogenicity in human epidemiology studies or experimental laboratory studies (Bingham and others 1979).

Substances known or suspected of being carcinogenic and contained in diesel oil in small amounts include benzo(a)pyrene and benzene. Benzo(a)pyrene (BaP), a potent carcinogen, is a PAH that also occurs at low levels in foods and in products of combustion, including cigarette smoke. Bioassays have found that the concentration of this single carcinogen can often serve as a guide in predicting carcinogenic potency, although other substances are also known to be involved (Bingham and others 1979).

There is sufficient evidence for the carcinogenicity of BaP in experimental animals: BaP has produced tumors in all of the nine species for which data have been reported following various methods of administration (DHHS 1985). It has both a local and systemic carcinogenic effect. EPA (1986e) has estimated the carcinogenic potency of BaP as 11.5 per mg/kg/day.

For benzene, another aromatic known to be present in diesel fuels, sufficient evidence exists for its carcinogenicity in experimental animals and in humans (DHHS 1985). Benzene has been shown to cause leukemia in workers with chronic exposure. The carcinogenic potency of benzene, however, is much less than that of BaP. EPA (1986f) has estimated the carcinogenic potency of benzene as 0.0445 per mg/kg/day. But benzene can occur at greater concentrations (about 29 ppm in No. 2 fuel oil) than BaP occurs in diesel oil. Consequently, the carcinogenic potencies of diesel oil have been estimated for this FEIS on the basis of the potencies of both benzene and BaP.

The cancer potency of diesel oil and the petroleum distillates in Esteron 99 was estimated from the potencies of both benzene and benzo(a)pyrene. Samples of diesel oil and fuel oil have been found to have a BaP content of only 26 ppb, but No. 2 heating oil (which may be subjected to cracking rather than straight-run distillation) can contain 600 ppb (Bingham and others 1979). The midpoint of this concentration range--313 ppb--has been used to calculate the carcinogenic potency of the petroleum distillates although most diesel fuels can be expected to have lower BaP contents. The content of benzene was assumed to be 28.5 ppm on the basis of an analysis of water extracts of No. 2 fuel oil by Anderson (1975), with corrections for solubility relationships. The resulting estimate of carcinogenic potency of the diesel oil carrier and Esteron 99's petroleum distillates is 4.9×10^{-6} (mg/kg/day)⁻¹. Seventy-four percent of this potency is due to the BaP component. Because this potency is about one thousandth of that of 2,4-D, it would not add significantly to the potency of the 2,4-D mixture.

Appendix D
Human Health Risk
Assessment
(Quantitative)

Section 4

Section 4

EXPOSURE ANALYSIS

INTRODUCTION

This section presents the methods and results of the herbicide exposure analysis. The first subsection contains the basic background information used in defining the exposure analysis methods. The terminology of herbicide use and the potential human exposure from that use are discussed.

The second subsection presents the methods used to estimate herbicide doses to workers and members of the general public. The methods used for determining lifetime doses to workers and the public to evaluate the risk of cancer are described. The second subsection also discusses the populations at risk in the vegetation management programs.

The third subsection gives the results of the routine and accidental dose calculations for workers and the public for each herbicide and the results of the lifetime dose estimation.

Some Helpful Terms

This subsection defines some of the terms used in the discussion of the exposure analysis methods and explains the relationship between the doses estimated in the analysis and the doses that might actually occur in future herbicide treatment operations. Other terms may be found in the Glossary.

Herbicide Characteristics

Most herbicides are packaged and sold by the manufacturer in liquid form as a concentrate with a specified number of pounds of active ingredient, usually between 1 and 10, per gallon of concentrate and with inert ingredients forming the remaining portion. Many of the herbicides also are marketed in the form of wettable powder and granular formulations.

Before herbicides are applied, they are mixed with a carrier, usually water, according to the manufacturer's label instructions for the particular treatment purpose and the desired application rate in pounds of active ingredient per acre. The amount of concentrate that produces the desired amount of active ingredient per acre treated normally is mixed with 10 to 15 gallons of carrier for every acre to be treated in aerial applications and with 50 to 100 gallons of carrier for every acre to be treated in ground applications. Herbicide concentrate, stored in 30- to 55-gallon drums, is prepared for application and then is transferred to application equipment by a mixer-loader, who uses a batch truck that has separate storage tanks for the carrier and for the herbicide mixture.

Herbicide application equipment is designed to cover the target plants with a minimum of off-target spray movement, called drift. Spray equipment nozzles are designed to produce medium to large droplets because smaller droplets tend to remain airborne and may drift with air currents away from the target vegetation. Despite the effectiveness of the application equipment used, some small fraction of the droplets may break up into smaller droplets that the wind could blow offsite. Hand application techniques, such as injection and hack and squirt, do not use sprays; thus, these techniques do not produce herbicide drift (see description of hand application in Section 2).

Exposure and Dose

Two primary conditions are necessary for a human to receive an herbicide dose that may result in a toxic effect. First, the herbicide must be present in the person's immediate environment so that it is available for intake. It must be in the air the person breathes, on the person's skin, or in the person's food or water. The amount of herbicide present in the person's immediate environment is the exposure level.

Second, the herbicide must get into the person's body by some route. If it is in the air, it may be inhaled into the air passages and lungs. If it is on the clothing or skin, it may penetrate the skin. The amount that moves into the body by any of these routes constitutes the dose.

Thus, although two people may be subjected to the same level of exposure--for example, two workers applying herbicide with backpack sprayers--one may get a much lower dose than the other by wearing protective clothing, using a respirator, or washing immediately after spraying. Exposure, then, is the amount of herbicide available to be taken in; dose is the amount that actually enters the body.

Potential Routes of Human Exposure

The potential routes of exposure to humans from herbicide treatment operations are illustrated in Figure 4-1. The routes of exposure considered in this risk assessment in estimating doses to workers and the public that might occur during routine operations or in the event of an accident are listed in Table 4-1 and are described below. Food items and drinking water sources that may lead to ingestion (dietary) exposures are listed in Table 4-2.

Potential Human Exposures from Routine Operations

The greatest doses to humans in routine herbicide applications are to workers who may be exposed while: (1) mixing and loading herbicide into application equipment, (2) applying herbicide to vegetation using ground-based equipment, or (3) supervising or monitoring aerial or groundbased herbicide applications. Use of protective clothing and equipment and adherence to proper cleanup procedures and label precautions in general lead to significant reductions in the doses of workers.

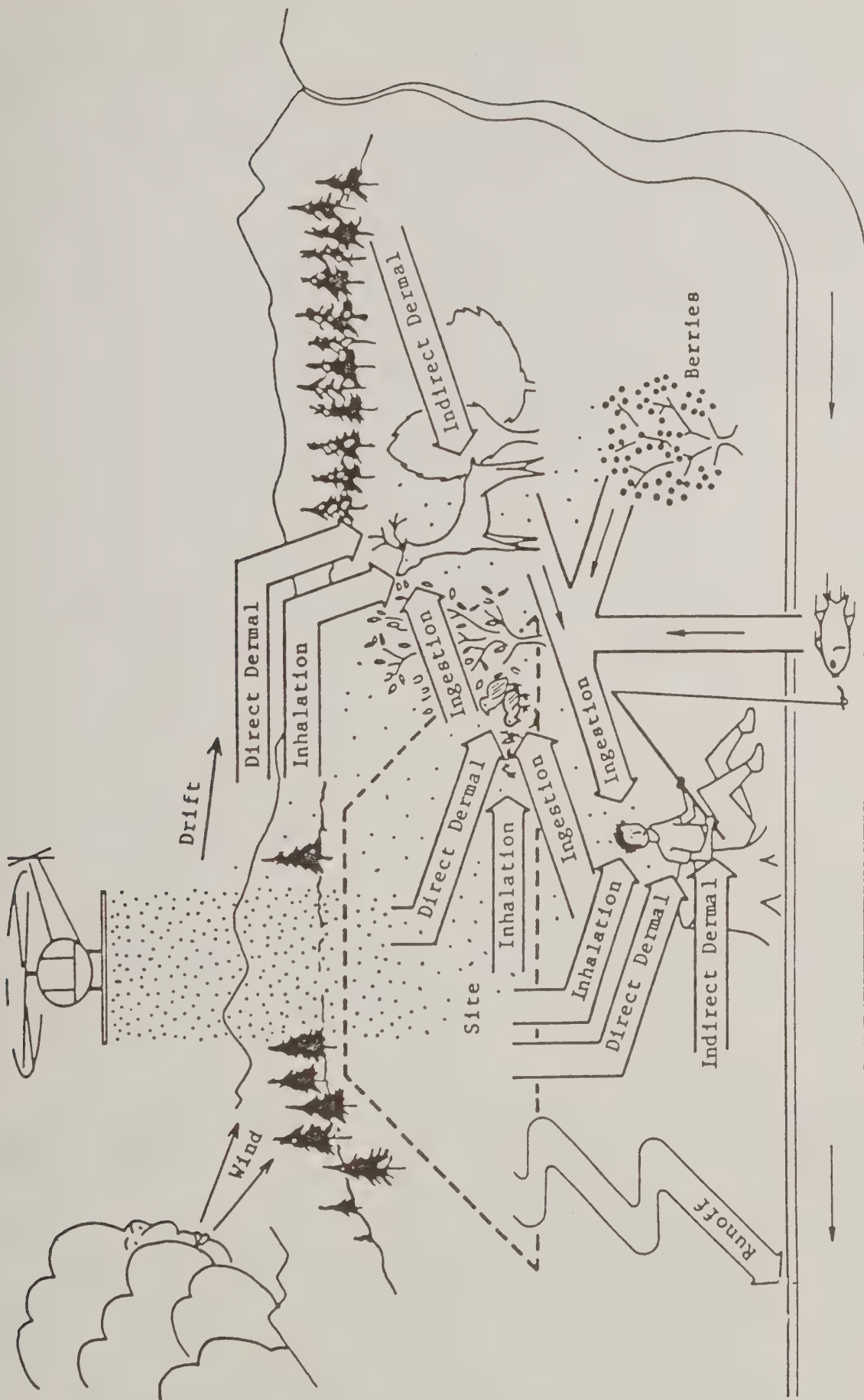


Figure 4-1 Routes of Exposure to Herbicides in Spraying Operations

Table 4-1
Routes of Exposure Considered in This Risk Assessment

	Doses from Direct Exposure	Doses from Indirect Exposure
<u>Routine</u>		
Workers	Total dose (based on field studies)	Dermal dose from reentry to treated area based on field data
General Public	Dermal dose ^a from drift (based on modeling)	Dermal dose from vegetation contact in drift area and from consuming food with residues ^b
<u>Accidental</u>		
Spraying	Dermal dose ^a to member of public directly sprayed	Worker vegetation contact dose from reentry to treated area immediately after spraying. Dose to member of public who walks through treated area and who eats directly sprayed food items ^b
Spills	Worker dermal dose from spill of concentrate or mixture on skin	Dose to member of public from drinking water contaminated by an herbicide spill

^aInhalation is negligible based on field study data.

^bSee Table 4-2 for diet items used in dose estimates.

The single most important source of exposure to persons who do not handle the herbicide containers or spray equipment in routine operations is from the drift off target of airborne herbicide spray droplets. Spraying only under favorable weather conditions and the use of spray equipment that limits the number of smaller spray droplets reduces the amount and extent of drift.

During routine operations, workers may be dermally exposed to an herbicide if the herbicide concentrate, mixture, or drifting spray droplets contact the skin or if the herbicide is brushed off of sprayed vegetation. Inhalation exposure may result from breathing without protective devices in the

Table 4-2

Dietary Exposures Estimated in This Risk Analysis

Scenario	Water	Food Items
<u>Routine</u>		
Realistic	Drift onto pond	Garden vegetables offsite Berries offsite Meat from a deer feeding offsite Meat from a game bird feeding offsite Fish caught in a pond receiving drift
Worst Case	Drift onto pond that is closer than realistic	Food items same as in realistic but closer to treatment unit
<u>Accidental</u>		
Spraying	Pond directly sprayed	Garden vegetables onsite Berries onsite Deer feeding in treated area Fish caught in pond directly sprayed Game bird directly sprayed
Spills	100- or 2,000-gal spill of mix in drinking water supply	

area of the drifting spray droplets or where there are vapors from a volatile herbicide. However, a variety of studies have shown that inhalation exposure is very small compared with dermal exposure. In this analysis, inhalation doses have not been estimated separately for workers; they are included with dermal doses in the estimated total worker doses based on herbicide levels in the urine of workers in field experiments.

Members of the general public who are within the area of drift of the smaller spray droplets may also receive dermal and inhalation exposure, but their exposures are relatively low compared to the exposures of workers directly involved in the spraying operations. Field studies of workers have consistently shown that inhalation exposure represents only a small part of the total exposure, so doses to the general public in this analysis have been calculated only for dermal and dietary routes (see description of worker studies later in this section).

Herbicide may be ingested by members of the general public from food containing herbicide residues. Food items such as garden vegetables, wild berries, or game animals may have received some level of herbicide from spray drift. Game animals may have fed on plants from the drift area. Ingestion exposure could also result from drinking water that has received herbicide drift or from eating fish from a body of water that has received herbicide drift.

Potential Human Exposures from Accidents

In the event of an accident, workers and members of the public may be exposed to much greater amounts of herbicide than they would under normal circumstances. Workers who spill the concentrate or some of the prepared spray mixture on their skin during mixing, loading, or spraying operations or who are doused when a transfer hose breaks would be dermally exposed. Workers or members of the public who are accidentally sprayed with herbicide because they are beneath a spray aircraft or are too close to a truck or backpack applicator would receive a dermal dose.

The dermal dose would depend on the concentration of herbicide in the spray mix, the area of the sprayed person's exposed skin, the extent to which the person's clothing absorbed herbicide (some clothing is water repellent, but other material would permit penetration of the herbicide to the skin), and the time that elapses before the person can wash. Indirect dermal (reentry) exposure may occur if workers or members of the public brush up against wet vegetation in the sprayed area.

Members of the public may accidentally be exposed to the herbicide by eating food or drinking water that has been directly sprayed. For example, members of the public may eat berries that have been directly sprayed, or they may eat meat from deer that have recently foraged on a sprayed site. Exposure to even higher levels of herbicide is possible if a container of herbicide concentrate were to break open and spill into a drinking water supply.

EXPOSURE ANALYSIS METHODS

Application Scenarios

To make reasonable estimates of the possible herbicide doses to workers and the public, a number of application scenarios are used that represent an array of likely treatment situations. Routine application scenarios were designed to provide a range of human dose estimates, from realistic to worst case, for normal operating conditions. Accidental-worst case scenarios--direct application, spills on the skin, and large spills into bodies of water--are used to estimate the highest doses that could ever be reasonably expected to occur. All but the lowest doses from all vegetation management projects conducted in the Pacific Northwest should fall within the range of doses predicted in these scenarios.

The scenarios specify those characteristics of each kind of herbicide application operation that determine human doses. For example, for workers involved in backpack operations, the number of work hours and the herbicide

application rate are used to determine their doses. For aerial applications, the number and size of the sites treated in a day's operation are used. To calculate doses to nearby residents who may eat a garden vegetable containing herbicide residue, it was necessary to estimate how much residue was on the vegetable and to specify how much of the vegetable was eaten.

The application scenarios were not intended to show what necessarily will happen as a result of a given treatment operation, but what could happen if all of the conditions specified in the scenario were met in the actual operations. For example, worker doses are based on actual dose levels found in field exposure studies in which no protective clothing or equipment was worn. If workers were to wear protective clothing and equipment during actual operations, their doses could be significantly lower than those estimated here. However, despite all precautions, workers present during treatment operations will be exposed to some extent.

Additional factors must be recognized when evaluating the likelihood of a member of the public receiving an herbicide dose. A forest user would receive a dose only in the immediate vicinity of the treatment area and only at the time of the herbicide application. However, because of the limited area of forest being treated and the public's restricted access and use, the possibility of this occurrence is slight. Likewise, a nearby resident would receive a dose as high as the one estimated in this analysis from eating garden vegetables with herbicide residue only if all of the following conditions were met:

1. The resident's garden was close enough to a particular treatment area to receive some level of herbicide drift.
2. The weather conditions on the day of treatment were such that the herbicide happened to drift offsite in the direction of the garden.
3. The resident ate the vegetable immediately after the herbicide residue landed on it.

A combination of factors makes the possibility of the resident receiving such a dose highly unlikely. First, most treatment areas are located considerably further from any residence than the distance assumed in this analysis--600 feet. Second, mitigation measures described in Section 2 reduce the likelihood of drift onto a garden, even if one happened to be nearby. Third, there is only a small possibility the resident would immediately pick and eat a garden vegetable that had herbicide residue from that operation.

Workers Doses from Routine Operations

Herbicide doses to workers involved in routine operations were estimated using eight herbicide application scenarios: four routine-realistic and four routine-worst case scenarios. For each application scenario, worker categories were chosen to represent the normal range of work activities in terms of potential herbicide exposure. Other categories of workers may

experience less exposure, but no category of workers in the field is expected to experience greater exposure than the types of workers considered in this analysis.

Doses to members of the public as a result of routine operations were estimated using three of the routine-realistic and three of the routine-worst case scenarios used to derive the worker doses. Again, other categories of the public may receive less exposure, but no one should receive more under normal operating conditions.

Worker Categories and Calculations in Routine Operations

Worker dose levels were extrapolated directly from worker doses determined by urine analysis in field studies of actual herbicide treatment operations. Because the field studies showed what dose levels are experienced in actual operations, they were considered the most appropriate basis for estimating the doses of Forest Service Region 6 and BLM herbicide applicators involved in the same or similar vegetation management practices. Those studies are discussed in the next subsection.

Dose estimates were scaled to the anticipated work hours and herbicide application rates specified in each of eight application scenarios.

Routine-realistic. To estimate routine-realistic worker doses, average dose levels found by urine analysis in field studies of workers exposed in spraying 2,4-D using the same application method were used. Nominal dose levels in mg/kg for workers in each category (see below) were derived from these average dose levels by dividing by the field study acreage and application rate.

Application rates for the routine-realistic dose scenarios are listed in Table 4-3.

The worker categories and scenarios used for estimating the routine-realistic worker doses included the following:

1. Doses to pilots, mixer-loaders, supervisors, and observers in a helicopter broadcast treatment of four 40-acre silviculture sites
2. Doses to applicators, mixer-loaders, and applicator/mixer-loaders in truck broadcast spraying of 12 acres (33 feet wide by 3 miles long) of roadway right-of-way
3. Backpack applicator doses in backpack spraying of a 6-acre facilities maintenance site by two applicators for 6 hours
4. Doses to applicators using hack-and-squirt and injection-bar methods in hand treatment of 3 acres by two applicators for 6 hours

Worker doses for each worker category were estimated by extrapolating from the average dose levels found in field studies of workers exposed to 2,4-D using the same application method. The following steps were involved:

Table 4-3

Application Rates Used for
Routine-Realistic and Routine-Worst Case Scenarios
(lb active ingredient/acre)

	Aerial		Backpack		Right-of-Way	
	Realistic	Worst Case	Realistic	Worst Case	Realistic	Worst Case
Amitrole	2.00	4.00	2.00	5.00	2.00	8.00
Asulam	2.40	3.34	1.20	3.34	2.40	5.00
Atrazine	3.75	4.00	3.00	4.00	3.00	8.50
Bromacil	0.00	0.00	4.00	10.00	4.00	10.00
2,4-D	2.50	4.00	2.00	4.00	2.50	4.10
2,4-DP	2.00	2.50	2.00	4.30	2.50	5.00
Dalapon	4.00	10.00	4.00	12.00	4.00	10.00
Dicamba	1.00	4.00	0.50	4.00	1.00	3.60
Diuron	0.00	0.00	4.00	6.00	4.00	16.00
Fosamine	3.00	12.00	3.00	11.50	4.00	10.70
Glyphosate	2.00	5.00	1.50	5.00	2.00	5.00
Hexazinone	2.50	3.00	1.12	3.00	2.50	6.00
Picloram	1.00	5.00	1.00	4.00	1.00	2.00
Simazine	4.00	5.00	2.00	4.60	2.00	4.60
Tebuthiuron	1.00	6.00	1.50	6.00	2.20	4.60
Triclopyr	2.00	8.00	2.00	8.00	2.00	8.00

1. The average dose observed in the 2,4-D field study was expressed in terms of dose per pound of active ingredient applied.
2. The acreage figure was used to determine the number of pounds of active ingredient used in the scenario by multiplying by the herbicide's typical application rate (listed in Table 4-3).
3. The herbicide-specific dose was determined by multiplying the pounds of herbicide applied by the dose of 2,4-D per pound of 2,4-D applied for that worker category in the field studies and then adjusting for the herbicide's dermal penetration rate. The dermal penetration rates used in the analysis were 6 percent for 2,4-D (Feldman and Maibach, 1974), 6.4 percent for 2,4-DP, 0.48 percent for picloram (Lavy et al., 1984), 5 percent for dicamba (Draper and Street, 1982), 0.1 percent for amitrole, and 10 percent for the other 11 herbicides (USDA, 1984).

The following example illustrates these steps. To calculate an applicator's exposure to dicamba during the Routine-Realistic Right-of-Way operation, the average 2,4-D dose to an applicator in the Nash et al. 1982 study (Table 4-4) of 0.012 mg/kg was divided by the average number of pounds of 2,4-D applied by the applicator, 70 pounds, to give an estimate of

the dose per pound of active ingredient applied, 1.7×10^{-4} mg/kg/lb. The next step was to calculate the total number of pounds of dicamba applied by multiplying the application rate for the scenario (Table 4-3) by the total number of acres treated in the operation, 12. For dicamba this would be 1.00 lb/acre x 12 acres = 12 lbs. applied. To calculate the dose, a correction factor was calculated for the difference in dermal penetration rates between dicamba (.05) and 2,4-D (.06), $.05/.06 = 0.83$. Finally, the dose was calculated as 1.7×10^{-4} mg/kg/lb x 12 lbs. x 0.83 = .0017 mg/kg (see Table B-5).

Routine-Worst Case. Routine-worst case worker doses were estimated for the same worker categories used in the routine-realistic scenarios. However, the site size, application rate, equipment type, meteorological conditions, and duration of exposure were set to those that would lead to the highest levels of exposure in herbicide treatment operations in the Region. Herbicide-specific dose levels in the routine-worst case scenarios were again derived from the worker field studies and weighted for application rate and hours exposed, but here the 95 percent upper confidence level of the field study doses was used for extrapolating to the nominal dose in mg/kg/hr for a 1-lb/acre application rate. Application rates used in the routine-worst case scenarios are listed in Table 4-3.

The worker categories and scenarios used for estimating routine-worst case worker doses included the following:

1. Doses to pilots, mixer-loaders, supervisors, and observers in fixed-wing broadcast spraying of a 400-acre site for range improvement
2. Doses to applicators, mixer-loaders, and applicator/mixer-loaders in truck broadcast spraying of 40 acres of transmission line right-of-way
3. Backpack applicator doses in backpack spraying of a 60-acre conifer release site by 14 applicators for 9 hours
4. Doses to applicators using hack-and-squirt and injection-bar methods in hand treatment of 9 acres by four applicators for 9 hours

The following example illustrates the calculation of the applicators exposure to dicamba during the Routine-Worst Case Right-of-Way operation. First, the upper bound of a 95 percent confidence interval for sprayer doses in the Nash et al. 1982 study was calculated as .0557 mg/kg, and divided by the average number of pounds applied, 70, to estimate the 95 percent upper confidence limit of dose per pound of active ingredient applied: 7.96×10^{-4} mg/kg/lb. Then the number of pounds of dicamba applied in the Routine-Worst Case scenario was calculated by multiplying the application rate (from Table 4-3) by the total number of acres treated per day, 40. For dicamba this would be 3.60 lb/acre x 40 acres = 144 lbs applied. To calculate the dose, the correction factor must be used to relate the dermal penetration rate of dicamba (.05) to that of 2,4-D (.06), $.05/.06 = 0.83$. Finally the dose was calculated as 7.96×10^{-4} mg/kg/lb X 144 lbs. X 0.83 = 0.095 mg/kg.

Field Studies of Worker Exposure to 2,4-D

Field studies of the exposures and resultant doses of workers using a variety of application equipment have been conducted on 2,4-D by Lavy et al. 1982, Lavy et al. 1984, Nash et al. 1982, and Franklin et al. 1982. Doses for each worker category found in the studies are listed in Table 4-4. Lavy et al. (1982) monitored three helicopter spray crews for worker exposure to 2,4-D, using portable air filters, denim patches, and urine analysis on two separate spraying dates; the first observing normal precautions, the second using special protective clothing and procedures. Nash et al. (1982) monitored exposure of workers to 2,4-D during aerial spraying in Washington and ground spraying in North Dakota under normal spray conditions (i.e., without special precautions).

(insert Table 4-4)

Lavy et al., (1984) investigated herbicide exposure to four spraying crews of 20 workers each, monitoring urine levels over two 5-day periods.

Franklin et al (1982) estimated worker exposure in pasture brush clearing operations in Saskatchewan using techniques similar to Lavy and coworkers. Urine samples were collected from personnel who conducted operations on 3 of 4 consecutive days.

The exposures used from the four worker exposure studies: (1) Franklin et al. 1982; (2) Lavy et al. 1982; (3) Nash et al. 1982; and (4) Lavy et al. 1984 are as follows:

<u>Scenario/Worker</u>	<u>Study</u>	<u>Dose (mg/kg)</u>
Small Aerial - Pilot	1, 2, 3	.0000755 (Weighted Aver.)
Small Aerial - Batchman	1, 2, 3	.000108 (Weighted Aver.)
Small Aerial - Supervisor	2	.00231
Small Aerial - Observer	2	.00049
Large Aerial - Pilot	1, 2, 3	.0002511 (Weighed aver., upper 97.5 percentile)
Large Aerial - Batchman	1, 2, 3	.0003201 (Weighted aver., upper 97.5 percentile)
Large Aerial - Supervisor	2	.0087 (Upper 97.5 percentile)
Large Aerial - Observer	2	.00155 (Upper 97.5 percentile)
Small Backpack - Sprayer	4	.075639
Large Backpack - Sprayer	4	.1895 (95 percentile)
Small R-O-W - Applicator	3	.012
Small R-O-W - Mixer Loader	3	.00681
Small R-O-W - A/M/L	3	.02
Large R-O-W - Applicator	3	.0557 (95 percentile)
Large R-O-W - M/L	3	.0179 (95 percentile)
Large R-O-W - A/M/L	3	.0445 (95 percentile)

All of the doses extrapolated from the worker studies above are based on work crews wearing ordinary work clothes and taking no special precautions against exposure.

Table 4-4
Doses of 2,4-D Measured in Exposure Studies for Each Worker Category

Investigator	Application Type and Rule	Equipment Used	Worker Category	Number of Workers	Method of Analysis	Doses	
						Average	Range
Lavy et al, 1982	Aerial, 2.2 Kg a.i./ha	Helicopter	Flagman	2			0.00119-0.00177 mg/kg/day
			Pilot	3	Denim Patches	0.00057 mg/kg	n.d.-0.0010 mg/kg
			Mechanic	3		0.0233	0.0233-0.0617
			Batchman	3		0.0448	0.0233-0.0911
			Supervisor	3		0.0167	n.d.-0.0005
			Observer	6		8 x 10 ⁻⁵	n.d.-0.0005
			Pilot	3	Urine	0.00248 mg/kg	0.00179-0.0557 mg/kg
			Mechanic	3		0.00068	0.00044-0.0136
			Batchman	3		0.00245	0.00215-0.0377
			Supervisor	3		0.00029	n.d.-0.0069
	Aerial	Helicopter, Special Precautions: Protective Coveralls, Gloves, Boots Hats, Goggles	Observer	6		0.00006	n.d.-0.0013
			Pilot	3	Denim Patches	3.3 x 10 ⁻⁵ mg/kg	n.d.-0.0001 mg/kg
			Mechanic	3		0.00577	0.0005-0.0162
			Batchman	3		0.01065	0.00016-0.0216
			Supervisor	3		0.0009	n.d.-0.0027
			Observer	6		0.0014	n.d.-0.0045
			Pilot	3	Urine	0.00854 mg/kg	n.d.-0.0237 mg/kg
			Mechanic	3		0.00301	n.d.-0.00516
			Batchman	3		0.01401	0.00053-0.0219
			Supervisor	3		0.00013	n.d.-0.00038
Nash et al., 1982	Aerial 585 kg a.i. Applied in 20 hrs.	4 Thrush Commanders 4 Grumman Ag-Cats 4 Pipers 1 Snow 1 Cesana	Observer	6		9 x 10 ⁻⁵	n.d.-0.00056
			Mixer- loader	6	Urine	0.0199 mg/kg	0.0008-0.0545 mg/kg
			Mixer/ loader-pilot	1		0.0180	
			Pilot	10		0.006 mg/kg	0.0013-0.0202 mg/kg

Ground 34 kg a.i. applied in 3.5 hrs.	Sprayers: 4 Pull-type 21 Self- propelled 10 cab 16 no cab	Sprayers	9	Urine	0.012 mg/kg	n.d.-0.0760 mg/kg
18 kg a.i. applied in 2.4 hrs.	Mixer- loaded	Mixer- loaded	7		0.0068	0.00165-0.0164
38 kg a.i. applied in 7.9 hrs.	Mixer/ loader- sprayer	Mixer/ loader- sprayer	8		0.020	0.0037-0.0442
Franklin et al., Aerial, 1982 1.68- 2.24 kg a.i./ha	Helicopter	Pilot Mixer	1 1	Urine	0.00322 mg/kg/day 0.013	
Lavy et al., 1984 (As cited in USDA, 1984) 1 gal. her- bicide/24 gal. water	Backpack	Operator	20	Urine	0.01752 mg/kg/day	n.d.-0.0903 mg/kg/day
Tordon 101-R (80% 2,4-D, 20% Picloram) Tordon 101-R	Injection bar	Operator	20	Urine	0.0019	n.d.-0.0095
Tordon 101-R	Hypohatchet	Operator	20	Urine	0.01696	n.d.-0.0866
Tordon 101-R	Hack and squirt	Operator	20	Urine	0.00576	n.d.-0.0451
Note: protective coveralls, gloves, boots hats, goggles used	Backpack Injection bar Hypohatchet Hack and squirt	Operator Operator Operator Operator Operator	20 20 20 20 20	Urine	0.0196 mg/kg/day 0.00086 0.0079 0.00244	0.0004-0.1175 mg/kg/day n.d.-0.0035 n.d.-0.0439 n.d.-0.0148

Why the Worker Dose Estimates Are Higher than Would Occur in Actual Operations

As described above, this risk assessment estimates two separate dose levels for each category of worker in routine operations, a realistic dose and a worst case dose. The realistic dose is an estimate of the average dose a worker should receive on a typical day during normal treatment operations. The realistic dose is based on combining average nominal doses from field studies with scenario conditions that are typical for Forest Service and BLM operations in the Pacific Northwest.

However, the realistic dose estimates are higher than those that would occur in actual operations for two reasons. First, the doses are based on field study doses of applicators who wore no special protective clothing or devices. Many of the field studies measured doses to workers both with and without protective gear, and the applicators in many of the proposed Forest Service and BLM operations will wear protective gear, but the lower doses of protected workers were not used in extrapolating to the doses estimated in this analysis. Second, during the field exposure studies, many of the less severe types of accidents occurred that could be termed operational errors. For example, pilots handled the transfer hoses and helped with the mixing and loading operations and, in one instance when a pump broke down, transferred spray mix by bucket to the spray tank. In both of these cases, these individuals received higher doses during that day's work than they would have otherwise. Nevertheless, their doses were used in deriving the average worker doses for that field study.

Total 2,4-D exposure to truck applicators via inhalation assuming an 8-hour day and a breathing rate of 29 L/min would be a maximum 0.03 mg versus a maximum 18 mg via dermal exposure according to data of Draper and Street (1982). Inhalation, therefore, constituted 0.17 percent of dermal exposure. Nigg and Stamper (1983) calculated inhalation exposure to be 0.03 percent of total body exposure for Florida airboat sprayers. Libich et al. (1984) in their study of right-of-way applicators using 2,4-D, 2,4-DP, and picloram found dermal exposure to be up to 50 times greater than exposure from inhalation.

The worst case estimates of worker doses in routine operations are extremely high for two reasons. First, the nominal dose levels from the field studies used for extrapolation are not the average doses seen but the dose at the upper limit of the 95-percent confidence interval as illustrated in Figure 4-2. This means that there is only 1 chance in 40 that a worker in the same field operation under the same conditions of terrain, weather, and equipment should receive a dose higher than the specified dose. Second, when this upper limit dose is combined with the assumptions of largest site size and highest application rate for dose extrapolation, extremely high doses are estimated that are unlikely to occur under true operational conditions. The probability of all of these events occurring at the same time, as discussed in Section 5, is less than 1 in 10,000. No workers are likely to receive a higher dose under routine operational conditions unless they are involved in one of the accidents described later in Section 4.

Because of the large number of actual field measurements, these extreme or routine-worst case estimates of doses to workers also take into account normal operational errors such as:

1. Errors of measurement during manufacturing and formulation
2. Errors of measurement during field mixing
3. Excessive swath overlap during application

Public Exposures and Doses from Routine Operations

Public Exposure Categories and Calculations for Doses from Routine Operations

Herbicide doses to the public potentially exposed to routine herbicide applications were estimated using six application scenarios. They are the same as the worker scenarios except hack-and-squirt and injection-bar methods were not included. The hand application scenario was excluded because no drift is involved and the chance that any other type of public contact with the herbicides might occur in these operations is negligible. In the remaining six scenarios, inhalation exposure was not estimated because none of the herbicides in question is a specific lung toxicant and because the worker field studies have consistently shown inhalation exposure to be an insignificant fraction of the total herbicide dose received (USDA, 1984). Only dermal field studies have consistently shown inhalation exposure to be an insignificant fraction of the total herbicide dose received (USDA, 1984). Only dermal and dietary routes of exposure were considered in this analysis.

The scenarios used for deriving routine-realistic public exposures and doses were:

1. Helicopter spraying of a single 40-acre silviculture site
2. Truck spraying of a 12-acre roadway right-of-way
3. Backpack spraying of a 6-acre facilities maintenance site by two applicators for 6 hours

The scenarios used for deriving routine-worst case public exposures and doses were:

1. Fixed-wing spraying of a 400-acre site for range improvement
2. Truck spraying of 40 acres of transmission line right-of-way
3. Backpack spraying of a 60-acre conifer release site by 14 applicators for 9 hours

Single Routes of Exposure. The following categories of exposure were estimated for each scenario: doses due to drift, vegetation contact by a hiker or berry picker, and the dietary exposures shown in Table 4-2.

Dermal dose estimates were derived from the estimated dermal exposure levels by assuming that 2 square feet of a person's skin was exposed and by adjusting for the dermal penetration rate of each herbicide. Ingestion dose estimates were made for the five specific food items and drinking water that receive herbicide residues shown in Table 4-2.

Multiple Routes of Exposure. In addition to estimating doses to the public from routine operations through the specific exposure routes described above, five categories of persons were assumed to receive doses simultaneously through a number of exposure routes: (1) a hiker, (2) a person who picks berries, (3) a hunter, (4) a fisherman, and (5) a nearby resident. Each of these persons was assumed to receive an herbicide dose that is the sum of the doses from several routes of exposure as shown in Table 4-5.

(insert Table 4-5)

It is extremely unlikely that a member of the public will receive simultaneous herbicide doses through more than two of the exposure routes described above. However, to ensure that no possible dose was omitted from the analysis, it was assumed that the hiker receives dermal exposure from drift as well as vegetation contact exposure from brushing against offsite plants that have received drift. The hiker also drinks water that has received herbicide drift. The berry picker receives the same dermal and drinking water exposure from drift as the hiker, but the berry picker is exposed to a higher level of vegetation contact exposure from brushing against plants that have received drift because of continuous contact with the berry plants. The berry picker also receives exposure from feeding on berries that have herbicide residues from drift.

Table 4-5

Multiple Routes of Exposure for Example People

Example People	Direct Dermal	Reentry Hiker	Reentry Berrypicker	Drinking Water	Eating			
					Berries	Vegetables	Deer	Bird Fish
Hiker	X	X		X				
Berrypicker	X		X	X	X			
Hunter	X	X		X			X	X
Fisherman	X	X		X				X
Nearby Resident	X	X		X		X		

X = Member of the public is exposed by this route.

The hunter is assumed to get the same dermal exposure, vegetation contact, and drinking water exposure as the hiker. In addition, the hunter is assumed to kill and eat a deer and a game bird that have been exposed and have fed on items in the area of herbicide drift. The fisherman receives the same doses as the hunter, except the fisherman eats fish taken from a pond that has received drift rather than eating a deer and a game bird. The nearby resident receives the same dermal exposure, vegetation contact, and drinking water exposure as the hunter, but the resident eats vegetables from a garden that has received herbicide drift.

Public Dose Estimation

Because no field studies existed on actual doses to the public comparable to those used for estimating worker doses, it was necessary to estimate public doses by modeling the transport and fate of the applied herbicides. Details of the transport and fate modeling are in the next subsection.

Surface herbicide residue levels were estimated using data from field studies of the drift and surface deposition of herbicides in aerial and ground-based spray operations. These empirical studies were used to calculate how much was deposited on people's skin and how much was deposited on food and vegetation, and in bodies of water.

The exposure models required input of expected distances to various sources of human exposure. Figures 4-3 through 4-8 illustrate the distances to sources of public exposure in each scenario. The distances were derived from an examination of currently used mitigation measures.

Example Calculation for a Routine-Realistic Right-of-Way Operation. Dose of atrazine to human from consuming 400 grams of fish.

The first step was to calculate the concentration per pound applied of the pesticide in a water body 6 inches deep. According to Table 4-7, the value of drift to water for the scenario is 0.0264 mg/ft^2 . The concentration in a 6-inch deep body of water per pound applied per acre is:

(insert Tables 4-6 and 4-7)

Table 4-6

Drift in mg/ft² Based on 1 lb/Acre
for Routine Scenarios

Aerial Scenario	Realistic	Worst Case
<u>Aerial Scenario</u>		
To Public and Crops	0.0020	1.0595
To Berries and Animals	0.1571	1.0595
To Water	0.6682	2.2491
<u>Right-of-Way Scenario</u>		
To Public and Crops	0.0043	0.0150
To Berries and Animals	0.0090	0.0150
To Water	0.0264	0.0353
<u>Backpack Scenario</u>		
To Public and Crops	0.0222	0.0428
To Berries and Animals	0.0341	0.0428
To Water	0.0588	0.0678

Table 4-7

Maximum Herbicide Concentrations in Drums and Batch Trucks

Herbicide	Pounds per Gallon Concentrate	Pounds per 50-Gallon Drum	Pounds per 2,000-Gallon Batch Tank
Amitrole	2.	100	800
Asulam	4.	200	668
Atrazine	4.	200	800
Bromacil	4.	200	400
2,4-D	4.	200	800
2,4-DP	6.	300	500
Dalapon	-- ^a	--	2,000
Dicamba	4.	200	800
Diuron	4.	200	640
Fosamine	4.	200	2,400
Glyphosate	3.	150	1000
Hexazinone	2.	100	600
Picloram	2.	100	1000
Simazine	4.	200	1000
Tebuthiuron	--	--	1200
Triclopyr	4.	200	1600

^aNot purchased in liquid formulation by the Forest Service or BLM.

$$\begin{array}{ccccccc}
 & & & & 3 & & \\
 & .0264 \text{ mg} & \frac{1}{\text{ft}} & \frac{1}{\text{ft}} & & \text{ppb} & \\
 & & \times & \times & \times & 1000 & = 1.86 \text{ ppb} \\
 & 2 & & & & 3 & \\
 & \text{ft} & 0.5 \text{ ft} & 28.3 \text{ kg/ft} & & \text{ppm} &
 \end{array}$$

The concentration of atrazine is adjusted for the lbs/acre applied for the scenario in Table 4-3:

$$1.86 \text{ ppb} \times 3.00 = 5.58 \text{ ppb}$$

The concentration in the fish is based on the bioconcentration factor of atrazine:

$$5.58 \text{ ppb} \times 5(\text{BCF}) = 27.9 \text{ ppb}$$

The dose to a 50-kg human based on consumption of 0.4 kg of fish is:

$$\frac{(27.9 \text{ ug/kg} \times 0.4 \text{ kg})}{50 \text{ kg}} = .223 \text{ ug/kg}$$

This value is given in Table B-17.

Why the Public Dose Estimates Are Higher than Would Occur from Actual Operations

The doses estimated for members of the general public are overestimates for a number of reasons. First, downwind concentrations on surfaces used to compute dermal exposure were those found on flat mylar deposition sheets.

The smaller spray particles in offsite drift tend to move around rather than impact on curved surfaces and therefore would have less of a tendency to adhere to a human's body. Second, no degradation of the herbicide is assumed to occur nor is the herbicide assumed to bind with any material, such as vegetation, so as to become biologically unavailable to humans. This would be an important factor in diminishing doses that may occur from any activity involving contact with treated vegetation.

The routine-worst case dose levels to the public can be considered the highest possible doses for routine spray operations because the doses are calculated in scenarios that combine many unlikely factors and events, including largest site size, highest application rate, least favorable weather conditions, and spray equipment most susceptible to offsite drift. No member of the public should get a dose that is any higher than the doses estimated in the routine-worst case scenarios except in the case of an accident.

Modeling Public Exposures and Doses

The following subsection presents a detailed discussion of the transport and fate modeling used in estimating herbicide doses to the public. Various sources for assumptions and methods of calculation were consulted (Dost, 1983; Crump, 1983; Simmons, 1983; USDA, 1984).

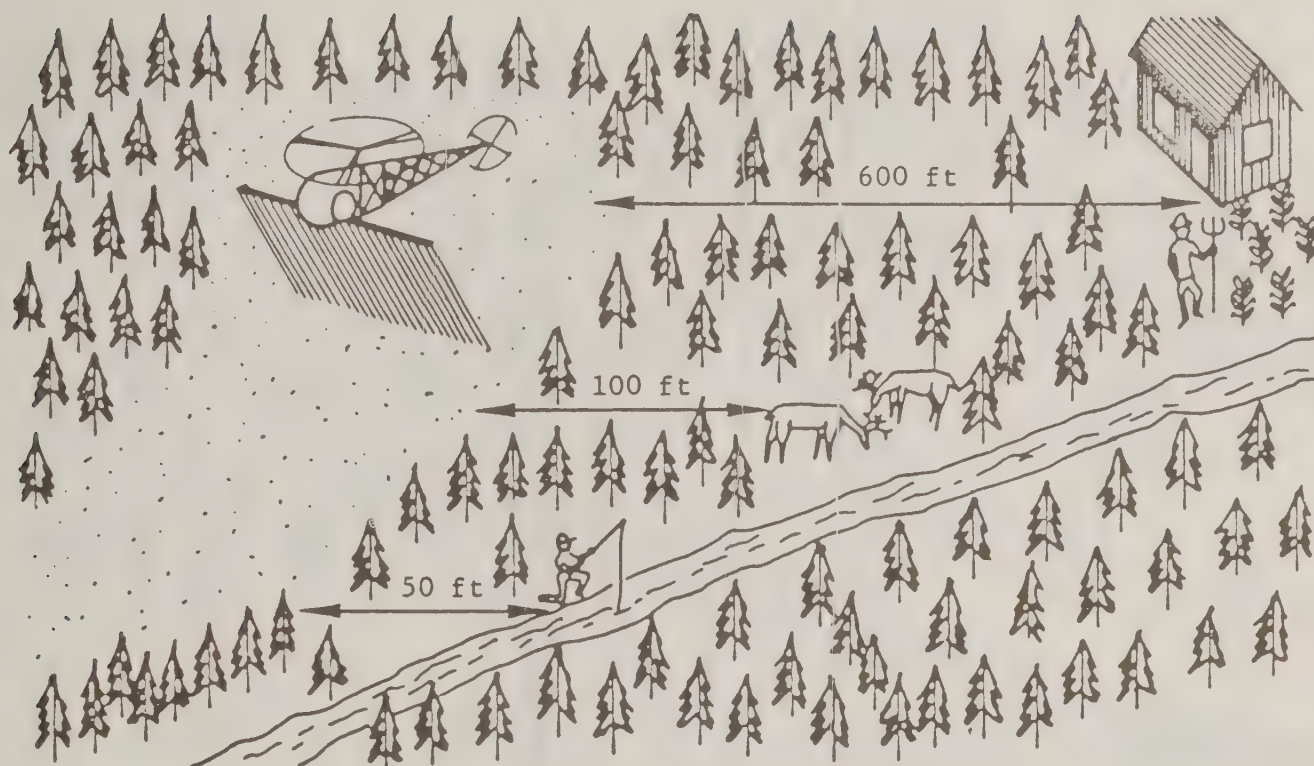


Figure 4-3 Routine-Realistic Aerial Scenario:
Helicopter Spraying of a 40-Acre Silviculture Site



Figure 4-4 Routine-Realistic Right-of-Way Scenario:
Truck Spraying of a 4-Acre Roadway Right-of-Way

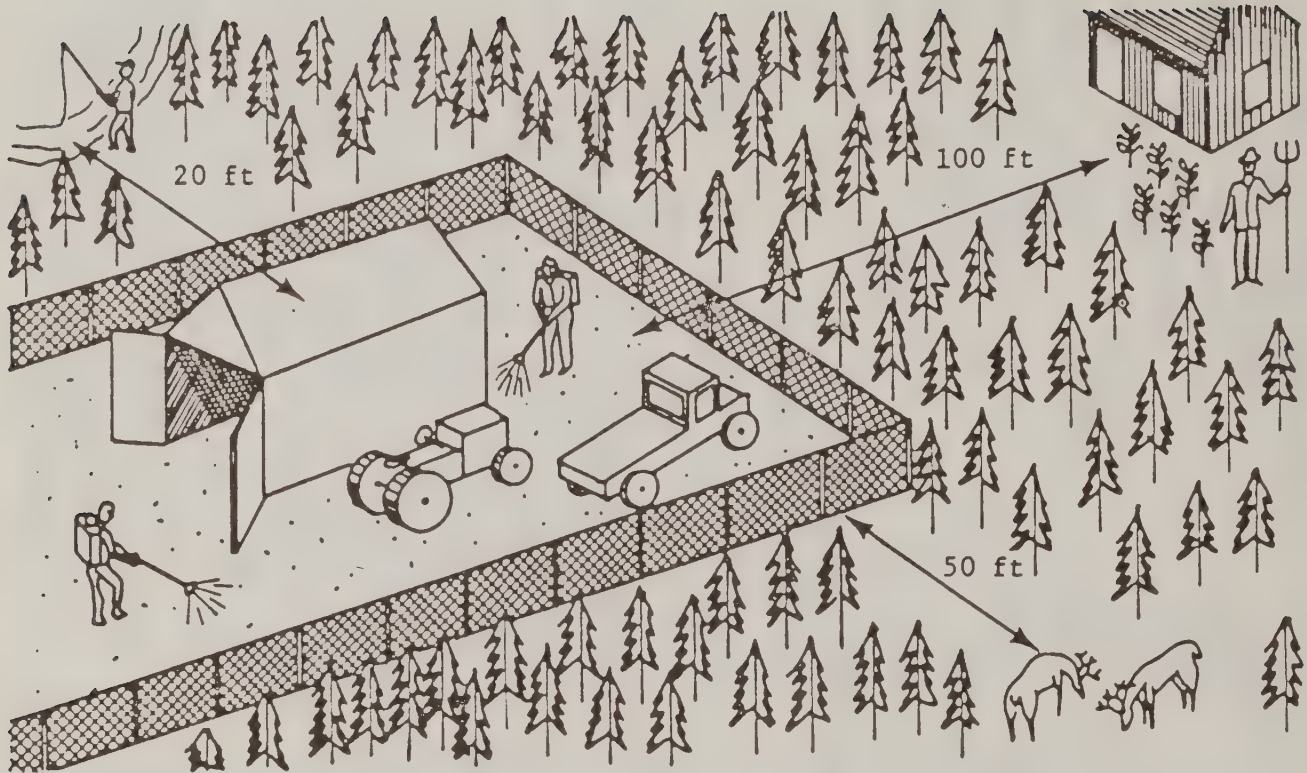


Figure 4-5 Routine-Realistic Backpack Scenario:
Treatment of a 6-Acre Facilities Maintenance Site

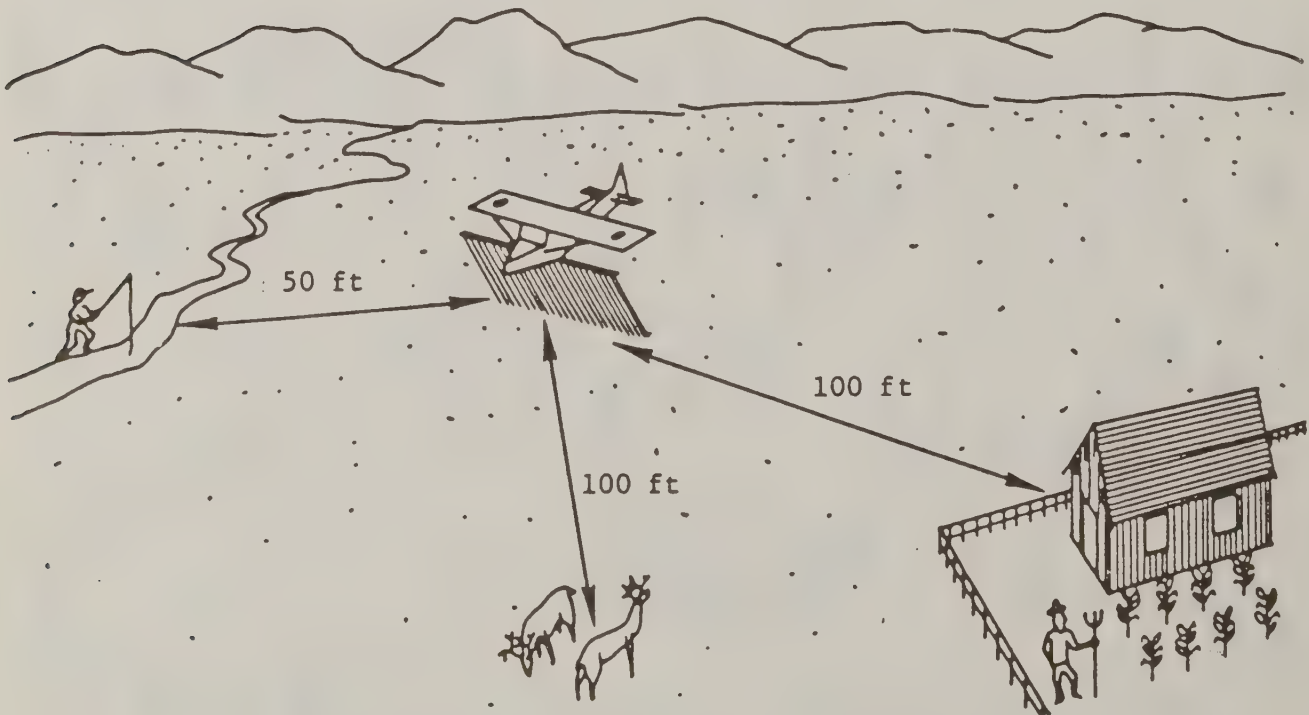


Figure 4-6 Routine-Worst Case Aerial Scenario
Fixed Wing Spraying of a 400-Acre Range Improvement Site



Figure 4-7 Routine-Worst Case Right-of-Way Scenario:
Truck Spraying of 40 Acres of Transmission
Line Right-of-Way



Figure 4-8 Routine-Worst Case Backpack Scenario:
Treatment of a 60-Acre Conifer Release Site

Spray Drift

The potential for herbicide sprays to drift onto adjacent lands or into nearby bodies of water was assessed based entirely on the results of empirical studies reported in the scientific literature. The analysis considered deposition on surfaces including exposed skin, water, game animals, and various classes of plants that may contribute directly or indirectly to the human diet.

Specific field studies were chosen to best represent the equipment and conditions appropriate for each scenario. Unfavorable conditions were chosen to show the degree of drift that could occur under the routine-worst case scenarios. Drift estimates for sprays applied in large range improvement projects were made based on the drift of 2,4-D from a fixed-wing aircraft (Miller, 1980). This test was conducted when winds averaged 9.5 mph. Mitigation measures specify no spraying if winds exceed 5 mph. Drift estimates for sprays applied in silvicultural projects were made based on drift of a dye tracer solution sprayed over a coniferous seed orchard by helicopter (Barry et al., 1983). The winds ranged from 4.5 to 9 mph. Drift of sprays applied by ground equipment was estimated based on a field test reported in Yates et al. (1978). In this test, glyphosate was sprayed by a ground sprayer when winds were 8.5 mph.

To facilitate use of the data from the various published field tests discussed above, a computer program was written to show how residues accumulate from multiple swaths (the long, narrow pattern of herbicide laid down by a broadcast sprayer such as an aircraft) and to correct for various application rates and swath widths. The program was then run to calculate deposition at selected representative distances for a nominal application rate of 1 pound per acre. The results are given in Table 4-6 for each of the six broadcast spray scenarios. The drift calculated for water bodies is intended to represent deposition at the edge of a minimum buffer strip (50 feet for aerial spraying and 20 feet for ground spraying).

Residues on Plants

Herbicide residues on plants on treated sites were estimated based on factors reported by Hoerger and Kenaga (1972). These factors were derived from a large number of studies, and they allow prediction of residues in parts per million (ppm) based on the application rate in pounds per acre. These residue estimates were calculated assuming no herbicide degradation, so they apply to conditions immediately after application. Following Hoerger and Kenaga (1972) the plants were classified into broad groups based on vegetative yield, surface-to-mass ratio, and plant interception factors. The residues estimated for each type of plant are intended to represent realistic yet relatively high estimates.

Offsite plant residues were calculated first for grasses based on the spray drift data discussed in the previous section and by using a regression equation given in Yates et al. (1978) to relate spray deposition on young wheat plants to that on sampling devices. The deposition was then estimated for other plant groups including berries and leafy vegetables by using the same relative factors given by Hoerger and Kenaga (1972), assuming that deposition on young wheat was approximately the same as deposition on range grass.

Herbicide doses to individuals were calculated assuming that they eat 400 grams (0.9 pounds) of contaminated berries or peas.

Residues in Water

Residues in water were calculated assuming that the water is only 6 inches deep, and that the herbicide spray drifts directly downwind to the water body over a minimum buffer distance. The buffer strips were assumed to be only 50 feet for aerial spraying and 20 feet for ground spraying. The actual residues in water would be less under more favorable spray conditions, at greater distances, or with deeper water bodies. For example, if the water were 2 feet deep then the residues would be only one-fourth of those calculated for this analysis. Dilution or degradation would also decrease residues. Herbicide doses to individuals were calculated assuming that they drink 1 liter of the maximally contaminated water.

Residues in Game Animals

Residues were calculated for two representative game animals: a 150-pound deer and a 0.25-pound game bird, such as a quail. The entire body surface area of the animal was assumed to be exposed to spray drift as shown in Table 4-6. Forty percent of the body surface was assumed to contact vegetation and thereby gain an additional average dermal residue level equal to that on the vegetation. Penetration of the herbicides through animal skin was assumed to be the same as through human skin.

The game animals were assumed to get an oral dose both by grooming and in their diet. The dose from grooming was assumed to amount to 29 percent of the nonabsorbed dermal dose for deer, and 40 percent for quail. The deer diet was assumed to consist of 2.45 kg of forage plants and 4 liters of water per day, both containing the herbicide. The quail diet was assumed to consist of 33 g of seed (grain) per day and 15 ml of water, both containing herbicide.

The concentration of herbicide in game meat was calculated by summing the animal's doses from both the dermal and oral routes of exposure and by assuming that 10 percent of that total dose was retained in the meat of the animal. This is similar to the method used in the exposure analysis of USDA (1984). Herbicide doses to humans were calculated by assuming that they eat 400 g of deer meat or 400 g of bird meat per day.

To illustrate the method used to determine human dietary exposures the following is the formula used to calculate the amount of herbicide in deer meat:

Variables used in the calculations:

WT = WEIGHT 150 lbs (68 kg) DRESS WT 120 lbs (54.4 kg)
BSA = BODY SURFACE AREA 2.6666 m,²
DFI = DAILY FOOD INTAKE 2.45 kg
BSCV = % BODY SURFACE CONTACTING VEGETATION 39%
BSG = % BODY SURFACE GROOMED 29%
WC = WATER CONSUMPTION 4 L/day
DPR = DERMAL PENETRATION RATE (CHEMICAL SPECIFIC)

DEER DERMAL DOSE:

- (1) DEPOSITION RATE X BSA = DIRECT DERMAL
ON SURFACE (mg/m²) EXPOSURE (DDE)(mg)
(% APPLICATION RATE)
- (2) DEPOSITION RATE ON X BSCV X BSA = INDIRECT DERMAL
VEGETATION EXPOSURE (IDE)(mg)
- (3) DDE + IDE = TOTAL DERMAL EXPOSURE (TDE)(mg)
- (4) TDE x DPR = DOSE ABSORBED THRU SKIN (DAS)(mg)

DEER INGESTION DOSE:

- (5) (DDE - DAS) X GSF = INGESTION VIA GROOMING (IVG)(mg)
- (6) FORAGE LEVEL X DFI = INGESTION VIA DIET (IVD)(mg)
- (7) CONCENTRATION X WC = INGESTION VIA DRINKING (IVDR)(mg)
IN WATER
- (8) IVG + IVD + IVDR = TOTAL INGESTION DOSE (TID)(mg)

(ASSUME INHALATION EXPOSURE INSIGNIFICANT)

- (9) DAS + TIS = TOTAL DOSE (TDOSE)(mg)
- (10) TDOSE X DOSE TO TISSUE = AMOUNT RETAINED
CONCENTRATION IN MEAT
RATIO 0.10 (RMEAT)(mg)
- (11) RMEAT/DRESS WT = (mg/kg) IN MEAT (PPMDEER)

Residues in Fish

Residues in fish were calculated assuming that the fish lived in and were caught from waters 6 inches deep, directly downwind of a treated site, with a minimum buffer strip of 20 feet for ground-based applications and 50 feet for aerial applications. For most of the herbicides considered in this analysis, which do not appreciably bioaccumulate, the concentration in fish were taken to be equal to the particular herbicide's concentrations in water. For the herbicides for which bioconcentration is likely to be greater--atrazine, diuron, and tebuthiuron--a bioconcentration factor was used. A bioconcentration factor of 10 was used for tebuthiuron, a value of 5 was used for atrazine, and a bioconcentration factor of 20 was used for diuron (Koeman et al., 1969). Doses to humans from eating fish containing herbicide were calculated assuming that 400 g are eaten daily.

Dermal Exposure

Dermal exposure due to drift was estimated by assuming that 2 square feet of skin were exposed (Dost, 1983) and the level of deposition on skin is the same as that found on the sampling sheets used in the drift monitoring studies. The dose was calculated as the deposited amount times the dermal penetration rate.

Indirect dermal exposure due to contact with foliage with surface residues of drifted herbicide was calculated by using the "unified field model" of Popendorf and Leffingwell (1982) and Popendorf (1985). This model was developed to estimate the possible doses and effects of insecticides on agricultural workers. The model was applied to estimate the relatively heavy exposures that could result from extensive foliage contact, such as that which would be experienced in berrypicking. The model takes into account:

1. The residue on foliage at any point in time after application (This analysis assumes no decay after initial application.)
2. A crop-specific residue transfer coefficient (cm^2/hr).
3. The exposure period in hours.
4. The dermal penetration rate for each herbicide and the body mass of a human (50 kg).

The residue transfer coefficient has been determined for a few agricultural situations. The value of $1,600 \text{ cm}^2/\text{hour}$ for this coefficient was used in this analysis to estimate doses to berry pickers. This value, derived from data collected for grape harvesting (Popendorf, 1985), represents a relatively high exposure situation. People engaged in activities involving less foliage contact, for example, tree planting, can be expected to receive doses that are considerably less. People who contacted foliage after the initial application also receive reduced doses due to degradation of the herbicides (see Table 4-9).

Dermal doses due to incidental contact with foliage, represented in the scenarios by vegetation contact for the hiker, were estimated by another method. Lavy et al. (1980) measured the level of 2,4,5-T on cloth patch samplers attached to a person who walked through a treated forest area. The residues were less than the detection limit of 0.01 mg per 100 cm² patch, but in this analysis a conservative assumption was made that the residues were at the detection limit. The area of clothing contacting foliage was assumed to be 40 percent of the total human surface area, and 10 percent of the total area was assumed to be bare skin contacting foliage. The same dermal penetration rates discussed previously were applied to bare skin, but the penetration through clothing was assumed to be 30 percent over a 6-hour period, based on work by Newton and Norris (1981).

Estimation of Doses to Workers and the Public from Accidents

The following scenarios were used to estimate the worst case doses that would result from the exposure to high amounts of herbicide that could occur in accidents.

1. Accidental Spraying. Members of the public are accidentally sprayed with herbicide because they are beneath a spray aircraft or too close to a truck or backpack applicator. (This dose would also apply to workers.) Indirect exposures to the same categories of people examined in the routine scenario are also estimated here. However, in the accidental-worst case spraying scenario, all items that they eat, drink, or brush against are sprayed at the full application rate, not just through drift.
2. Spills. Members of the public receive herbicide exposure via drinking water when a load of herbicide mixture is spilled or when a container of herbicide concentrate breaks open and spills into a drinking water supply. Workers spill concentrate or prepared spray mixture on their skin during mixing, loading, or spraying operations; or are doused when a transfer hose breaks.

Accidental dermal doses were derived from modeling the dermal penetration of herbicide concentrate or mixture for direct exposures. Accidental ingestion doses were estimated by modeling the dilution of herbicide concentrate or mixture in a body of water of a given size.

To calculate the dose to a person directly sprayed at the full per-acre application rate, the worst case application rates shown in Table 4-3 were converted to mg/ft². It is assumed that 2 square feet of human skin is exposed (Dost, 1983).

For example, the application rate of dicamba in the routine-worst case aerial application scenario is 4.0 lb a.i./acre (Table 4-3). This can be converted to kg/ha by the use of a conversion factor:

$$4.0 \text{ lb. a.i./acre} \times 1.12 \frac{\text{kg/ha}}{\text{lb/acre}} = 4.48 \text{ kg. a.i./ha}$$

$$4.48 \text{ kg. a.i./ha} \times 10^6 \frac{\text{mg}}{\text{kg}} \times 0.0001 \text{ ha/m}^2 \times 0.093 \frac{\text{m}^2}{\text{ft}^2} = 41.6 \text{ mg/ft}^2$$

If a square foot of skin was exposed on a 50 kg person, then the surface deposit in mg/kg would be 41.6 mg/ft². The absorbed dose must consider the dermal penetration rate: 1.66 mg/kg x 0.05 (dermal penetration rate) = 0.083 mg/kg absorbed. This is equivalent to 83 ug/kg, as given in Table 4-14.

Reentry exposure to the general public is estimated assuming an individual walks through a treated area after an operation has been completed, even though the area is posted. Reentry exposure is also calculated for an individual who picks berries for 4 hours in a treated area, even though the area is posted. Accidental dietary exposure is derived by assuming an individual eats food items that have been directly sprayed rather than food items receiving only spray drift, or eats meat from animals that have fed directly on sprayed vegetation or fish taken from directly sprayed water bodies or drinking water from those water bodies.

An individual receives an accidental ingestion exposure resulting from a major spill by drinking water from a pond or a reservoir that has been contaminated by a dump of 100 gallons of herbicide mix as from a helicopter, or 2,000 gallons of spray mix from a batch truck. Two thousand gallons is approximately the largest amount of spray mix that might be carried by a tank truck supplying a large aerial spraying operation. One hundred gallons is approximately the largest load that can be carried by the types of helicopters currently used in the Pacific Northwest. The maximum herbicide concentrations in drums and batch trucks are shown in Table 4-7. The pond is assumed to be 1 acre in area and 4 feet deep, and to have no inflow or outflow. The reservoir is assumed to be 16 acres in area and 8 feet deep. A person is assumed to drink 1 liter of water after complete mixing has occurred.

Direct dermal exposures were calculated for spills of 0.5 liter of herbicide concentrate (if liquid concentrates are used) or 0.5 liter of the most concentrated spray mixture. The person exposed during the spill is assumed to weigh 50 kg, and most of his surface area (0.8 m^2 or 8.6 ft^2) is thoroughly wetted by the solution. Denim fabric commonly used in clothing retains about 57.5 ml of solution per square foot (Weeks, 1985), and absorption of herbicide through the cloth was calculated as before, based on Newton and Norris (1981). However, 20 percent of the solution was assumed to wet bare skin. A spill resulting in this exposure could result from broken hoses, spilled containers, or emergency and accidental dumps by helicopters.

Estimation of Lifetime Doses to Workers and the Public

Doses used in the cancer risk analysis for 2,4-D, 2,4-DP, picloram, amitrole, asulam, bromacil, and glyphosate (discussed in Section 5) were derived by combining available information on the number of days per year an individual worker may spray an herbicide using a particular application method, and estimates of the expected daily dose and the number of years of employment. Expected daily doses were calculated assuming that the worst case dose is experienced 5 percent of the time and the realistic dose 95 percent of the time, in all routine scenarios. The realistic cases assume that workers are employed in pesticide application for 5 years, and the worst cases assume 20 years employment in herbicide applications.

Average numbers of exposures per lifetime were used with expected daily doses for each scenario to derive realistic lifetime doses. Extreme lifetime doses were derived by multiplying expected daily dose levels estimated in worker scenarios by estimates of the highest number of days a worker is likely to be engaged in the particular type of application method. Exposures per lifetime in the realistic scenarios were estimated to be the following: aerial, 30; right-of-way, 45; backpack, 50; and hand application, 70. For the routine-worst case scenario doses, the number of exposures per lifetime were: aerial, 288; right-of-way, 416; backpack, 440; and hand application, 480.

Lifetime exposures to the public for the five herbicides were derived by assuming a realistic estimate would be a single exposure per lifetime in each of the public exposure scenarios, and a high estimate would be one exposure per year for 30 years. The exposure levels derived in the realistic and extreme public scenarios and on accidental spraying and spills were multiplied by 1 for realistic lifetime and 30 for extreme lifetime doses.

Effect of Body Size on Exposure

All doses estimated in the exposure analysis were calculated for a representative 50-kg person. This weight was chosen to represent an adult of less than average weight, so that doses to adults would be calculated in a conservative manner. Doses for a larger person would be less in terms of mg per kg body weight. For example, a 70-kg person would receive approximately 25 percent more herbicide than a 50-kg person by dermal exposure, because of his greater surface area. A 70-kg person would also receive on average about 25 percent more herbicide by dietary exposure routes, because both body surface area and metabolism are approximately proportional to body weight raised to the 2/3 power:

$$\frac{(70)^{2/3}}{(50)^{2/3}} = 1.25$$

However, a 70-kg person also has a body weight greater than a 50-kg person, by a greater factor:

$$\frac{70}{50} = 1.4$$

The combined effect of these two factors is that a 70-kg person will receive a dose in mg/kg that is only 89 percent as great as for a 50-kg person.

Conversely, smaller people can be expected to receive greater doses in terms of mg per kg body weight. A 20-kg child will receive only about 54 percent as much herbicide as a 50-kg person, but his weight is only 40 percent as great. The net effect is that a 20-kg child will receive a dose that is 36 percent greater in terms of mg/kg than it would be for a 50-kg person.

It should be noted that small children may, in some cases, be among the more sensitive individuals.

Table 4-8 illustrates the effect of body size on expected dose. The table shows doses for a 20-kg child and 50- and 70-kg adults for each route of exposure in the extreme aerial application scenario.

Table 4-8

Effect of Body Size on Dose:
2,4-D Aerial Routine-Worst Case Scenario
(doses in micrograms/kg)

Exposure Route	20-kg Child	50-kg Adult	70-kg Adult
Drift, Dermal	13.86	10.19	9.07
Veg. Contact, Hiker	0.20	0.15	0.13
Veg. Contact, Picker	35.72	26.27	23.38
Drinking Water	17.25	12.68	11.29
Eating Berries	14.18	10.42	9.28
Eating Vegetables	28.35	20.85	18.55
Eating Deer Meat	2.16	1.59	1.41
Eating Game Bird	9.20	6.77	6.02
Eating Fish	6.90	5.07	4.51

Time Dependence of Dermal Exposure Due to Vegetation Contact

Herbicide residues on plant surfaces decline over time as a result of absorption by the plant, degradation, volatilization, and washing by rainfall. After herbicide sprays dry on plant surfaces they cannot be completely rubbed off because of binding to the plant surface materials. Consequently, persons entering a treated area a short time after spraying are likely to receive dermal doses much smaller than the conservative doses calculated in this analysis. However, specific data were not available for most of the 16 herbicides regarding persistence on plant surfaces. The most appropriate data would be measurements of dislodgeable residues, but this type of data was not available for the herbicides. In most cases, measurements of total plant residues over time were available, so this data has been used to calculate degradation rates in those cases where surface measurements were unavailable. Degradation rates calculated in this way should be considered minimum degradation rates for dislodgeable residues, because the residues that were measured in deriving the data may have been largely or entirely unavailable for dermal exposure through vegetation contact. Degradation rates for the 16 herbicides were determined using the following references:

(1) amitrole	Ghassemi et al. (1981)
(2) asulam	Gortz and Van Oorschot (1984)
(3) atrazine	Montgomery and Freed (1961)
(4) bromacil	WSSA (1983)
(5) 2,4-D	USDA (1984)
(6) 2,4-DP	USDA (1984)
(7) dalapon	USDA (1984)
(8) dicamba	USDA (1984)
(9) diuron	Leonard et al. (1975)
(10) fosamine	Ghassemi et al. (1981)
(11) glyphosate	Newton and Dost (1981)
(12) hexazinone	USDA (1984)
(13) picloram	Bovey et al. (1967)
(14) simazine	Ghassemi et al. (1981)
(15) tebuthiuron	Bovey et al. (1978)
(16) triclopyr	USDA (1984)

Table 4-9 shows the dermal exposures calculated for a hiker and a person picking berries for 4 hours on a treated site. The table shows the doses at the first day and also after 30 and 90 days. The doses decline dramatically even with these minimum rates of degradation. In the case of bromacil, no degradation rate was found in the available literature, so a minimal degradation rate corresponding to a half life of 60 days was used. The actual rate of degradation is likely to be greater.

Even the 90-day time period is considerably less than the minimum period between treatment for site preparation and reentry for tree planting. Vegetation contact for tree planters is also much less than for berry pickers, so the maximum dose for planters will be significantly less.

EXPOSURE ANALYSIS RESULTS

This subsection presents the results of the exposure analysis. Doses to workers and the public estimated for routine operations and for accidents are summarized and discussed. Complete dose estimates are presented in Attachment B.

Doses to Workers

Realistic Worker Doses in Routine Operations

Routine-realistic worker doses are summarized in Table 4-10. No worker in any of the realistic scenarios receives a dose of any herbicide greater than 1.0 mg/kg. All backpack workers receive doses greater than 0.1 mg/kg except those using amitrole, dicamba, and picloram. Helicopter mixer-loaders receive atrazine, dalapon, and simazine doses greater than 0.1 mg/kg, but all other doses in the realistic aerial scenario for mixer-loaders, pilots, supervisors, and observers are less than 0.1 mg/kg. Bromacil, 2,4-DP, diuron, fosamine, and triclopyr doses to hack-and-squirt applicators are greater than 0.1 mg/kg. All other hand application doses are less than 0.1 mg/kg.

Table 4-9

Doses Due to Vegetation Contact on a Treated Site
(micrograms/kg)

Herbicide	Degradation Rate per Day	Hiker			Berrypicker		
		Day 1	Day 30	Day 90	Day 1	Day 30	Day 90
Amitrole	0.0866	12.0	0.9	0.0	7168.0	533.5	3.0
Asulam	0.0110	4.2	3.0	1.6	2508.8	1803.6	932.2
Atrazine	0.0621	15.0	2.3	0.1	8960.0	1390.6	33.5
Bromacil	0.0116	18.0	12.7	6.3	10752.0	7592.0	3785.2
2,4-D	0.0431	2.0	0.6	0.0	1225.7	336.4	25.3
2,4-DP	0.0431	3.0	0.8	0.1	1792.0	491.8	37.0
Dalapon	0.0542	18.0	3.5	0.1	10752.0	2115.1	81.8
Dicamba	0.0578	4.8	0.8	0.0	2867.2	506.3	15.8
Diuron	0.2740	35.9	0.0	0.0	21504.0	5.8	0.0
Fosamine	0.0990	7.2	0.4	0.0	4300.8	220.5	0.6
Glyphosate	0.0495	3.0	0.7	0.0	1792.0	405.9	20.8
Hexazinone	0.0584	7.2	1.2	0.0	4300.8	745.9	22.4
Picloram	0.0693	0.0	0.0	0.0	18.6	2.3	0.0
Simazine	0.0455	2.8	0.7	0.0	1648.6	421.0	27.5
Tebuthiuron	0.0834	4.8	0.4	0.0	2867.2	234.9	1.6
Triclopyr	0.3120	4.8	0.0	0.0	2867.2	0.2	0.0

Table 4-10

Worker Doses for Routine-Realistic Scenarios

Herbicide Application	Aerial				Truck				Hand	
					App					
Hack/ Bar	Pilot	Mix/L	Sup	Obs	Backpack	Appl	Mix/L	M/L	Squirt	Inj
Amitrole	0	0	-1	-1	1	-1	-1	-1	0	0
Asulam	2	2	1	1	3	1	1	2	--	--
Atrazine	2	3	2	1	3	2	2	2	--	--
Bromacil	--	--	--	--	3	2	2	2	3	2
2,4-D	2	2	1	0	3	1	1	1	2	2
2,4-DP	2	2	1	0	3	1	1	1	3	2
Dalapon	2	3	2	1	3	2	2	2	--	--
Dicamba	2	2	1	0	2	1	1	1	2	2
Diuron	--	--	--	--	3	2	2	2	3	2
Fosamine	2	2	1	1	3	2	2	2	3	2
Glyphosate	2	2	1	1	3	1	1	1	--	--
Hexazinone	2	2	1	1	3	1	1	2	--	--
Picloram	0	1	0	-1	1	0	0	0	1	1
Simazine	2	3	2	1	3	1	1	1	--	--
Tebuthiuron	2	2	1	0	3	1	1	2	--	--
Triclopyr	2	2	1	1	3	1	1	1	3	2

Legend

4	10.0 mg/kg
3	1.0 mg/kg (less than 1 milligram/kg)
2	0.1 mg/kg
1	0.01 mg/kg
0	0.001 mg/kg (less than 1 microgram/kg)
-1	0.0001 mg/kg
-2	0.00001 mg/kg

-- herbicide not used in this scenario

Table 4-11

Worker Doses for Routine-Worst Case Scenarios

Herbicide	Aerial				Backpack	Truck			Hand Application		
	Pilot	Mix/L	Sup	Obs		Appl	Mix/L	App M/L	Hack/ Squirt	Inj	Bar
Amitrole	1	1	1	0	2	1	1	1	2	1	
Asulam	3	3	2	2	4	3	3	3	--	--	
Atrazine	3	3	3	2	4	3	3	3	--	--	
Bromacil	--	--	--	--	4	3	3	3	4	3	
2,4-D	3	3	2	2	4	3	2	2	4	3	
2,4-DP	3	3	2	1	4	3	2	3	4	3	
Dalapon	4	4	3	2	4	3	3	3	--	--	
Dicamba	3	3	2	2	4	2	2	2	3		
Diuron	--	--	--	--	4	3	3	3	4	3	
Fosamine	4	4	3	2	4	3	3	3	4	3	
Glyphosate	3	4	3	2	4	3	3	3	--	--	
Hexazinone	3	3	2	2	4	3	3	3	--	--	
Picloram	2	2	1	1	3	1	1	1	2	1	
Simazine	3	4	3	2	4	3	3	3	--	--	
Tebuthiuron	4	4	3	2	4	3	3	3	--	--	
Triclopyr	4	4	3	2	4	3	3	3	4	3	

DOSE LEVELS

4	10.0 mg/kg
3	1.0 mg/kg
2	0.1 mg/kg
1	0.01 mg/kg
0	0.001 mg/kg
-1	0.0001 mg/kg
-2	0.00001 mg/kg

-- herbicide not used in this scenario

Worst Case Worker Doses in Routine Operations

In the routine-worst case worker application scenarios (summarized in Table 4-11), doses to aerial supervisors and observers, right-of-way applicators, and injection-bar applicators are all less than 1.0 mg/kg. Doses of dalapon, fosamine, tebuthiuron, and triclopyr exceed 1.0 mg/kg for pilots and mixer-loaders in the aerial scenario. Doses of simazine and glyphosate to mixer-loaders also exceed 1.0 mg/kg. All herbicide doses to backpack workers exceed 1.0 mg/kg except for amitrole and picloram. Hack-and-squirt applicator doses of bromacil, 2,4-DP, diuron, fosamine, and triclopyr all exceed 1.0 mg/kg. All other doses are less than 1.0 mg/kg.

Doses to the Public

Doses via Individual Exposure Routes

Doses to the public via specific exposure routes in the three routine-realistic scenarios are shown in Attachment B. In none of the routine-realistic scenarios does the public receive a dose greater than 0.01 mg/kg (10 micrograms/kg) for any of the 16 herbicides through any single exposure route. Doses are lowest (less than 0.006 micrograms/kg) to the hiker who contacts vegetation with herbicide residues. Doses are highest for the ingestion routes of exposure, particularly drinking water and eating berries or garden vegetables.

Doses to the public via individual exposure routes in the routine-worst case scenarios also are shown in Attachment B. No dose to the public is greater than 0.01 mg/kg for any chemical through any exposure route in either the truck (right-of-way) or backpack scenarios. Highest public doses occur in the routine-worst case aerial scenario, in particular, for the berrypicker contacting vegetation, where the berrypicker dose is slightly higher than 0.1 mg/kg (100 micrograms/kg) for dalapon and fosamine. It is less than 0.1 mg/kg for all the other herbicides.

Doses via Multiple Exposure Routes

Doses for people who receive combined herbicide doses of each of the 16 herbicides through the various exposure routes outlined in Table 4-5 are listed for the three routine-realistic scenarios in Table 4-12, and for the three routine-worst case scenarios in Table 4-13.

(insert Tables 4-12 and 4-13)

In no instance do any of the combined doses of any of the herbicides in the routine-realistic scenarios exceed 0.1 mg/kg. Those that exceed 0.01 mg/kg (berrypickers for atrazine, dalapon, and simazine; fishermen for atrazine) are all lower than 0.015 mg/kg (see Attachment B).

No representative member of the public receives a dose of any herbicide higher than 0.01 mg/kg in the routine-worst case right-of-way scenario. Dalapon and fosamine doses to berrypickers exceed 0.01 mg/kg in the routine-worst case backpack scenario. Highest public doses range from greater than 0.01 mg/kg to less than 0.3 mg/kg for combined public doses in the routine-worst case aerial scenario for all 16 herbicides. Highest public doses are from dalapon and fosamine.

Table 4-12
Doses to Example People in
Routine-Realistic Exposure Scenarios

Herbicide	Aerial				Truck (row)				Backpack						
	Hiker	Berry- picker	Hunter	Fisher- man	Nearby Resident	Hiker	Berry- picker	Hunter	Fisher- man	Nearby Resident	Hiker	Berry- picker	Hunter	Fisher- man	Nearby Resident
Amitrole	1	1	1	1	1	-1	0	0	0	0	0	0	0	0	0
Asulam	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0
Atrazine	1	2	1	2	1	0	0	0	0	0	0	1	0	1	1
Bromacil	-	-	-	-	-	1	1	1	1	1	1	1	1	1	1
2,4-D	1	1	1	1	1	0	0	0	0	0	0	1	0	0	0
2,4-DP	1	1	1	1	1	0	0	0	0	0	0	1	0	0	0
Dalapon	1	2	1	1	1	0	0	0	0	0	0	1	1	0	1
Dicamba	0	1	1	1	1	-1	0	-1	-1	0	-1	0	0	-1	0
Diuron	-	-	-	-	-	0	0	0	1	0	0	1	1	1	1
Fosamine	1	1	1	1	1	0	0	0	0	0	0	1	0	0	1
Glyphosate	1	1	1	1	1	0	0	0	0	0	0	1	0	0	0
Hexazinone	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0
Picloram	0	1	1	1	1	-1	-1	-1	-1	0	-1	0	0	0	0
Simazine	1	2	1	1	1	0	0	0	0	0	0	1	0	0	0
Tebuthiuron	0	1	1	1	1	0	0	0	0	0	0	1	0	0	0
Triclopyr	1	1	1	1	1	0	0	0	0	0	0	1	0	0	0

DOSE LEVELS

- not used
2 0.1 mg/kg
1 0.01 mg/kg
0 0.001 mg/kg
-1 0.0001 mg/kg

Table 4-13
Doses to Representative Members of the Public in
Routine-Worst Case Exposure Scenarios

Herbicide	Aerial			Truck (row)			Backpack			
	Hiker	Berry- picker	Hunter	Fisher- man	Nearby Resident	Hiker	Berry- picker	Hunter	Fisher- man	Nearby Resident
Amitrole	2	2	2	2	2	0	1	0	0	1
Asulam	2	2	2	2	2	0	1	0	0	1
Atrazine	2	2	2	2	2	0	1	1	1	1
Bromacil	-	-	-	-	-	0	0	0	0	1
2,4-D	2	2	2	2	2	0	1	0	0	1
2,4-DP	2	2	2	2	2	0	1	0	1	1
Dalapon	2	3	2	2	3	1	1	1	1	1
Dicamba	2	2	2	2	2	0	0	0	0	1
Diuron	-	-	-	-	-	1	1	1	1	1
Fosamine	2	3	3	3	3	1	1	1	2	1
Glyphosate	2	3	2	2	2	0	1	0	1	1
Hexazinone	2	2	2	2	2	0	1	0	0	1
Picloram	2	2	2	2	2	0	0	0	1	0
Simazine	2	3	2	2	2	0	1	0	1	1
Tebuthiuron	2	3	2	3	2	0	1	0	1	1
Triclopyr	2	3	2	2	3	0	1	1	1	1

DOSE LEVELS

3 1.0 mg/kg
2 0.1 mg/kg
1 0.01 mg/kg
0 0.001 mg/kg
-1 0.0001 mg/kg

Accidental Doses to Workers and the Public

Doses from Accidental Spraying

Doses to workers and members of the public from accidental spraying are listed for the 16 herbicides in Table 4-14. The table lists doses in micrograms/kg rather than mg/kg (1 mg = 1,000 micrograms). Doses exceed 1 mg/kg for berrypicker dermal exposure (reentry) for bromacil, dalapon, diuron, and fosamine. Doses of diuron from eating fish exceed 1 mg/kg. All other doses are less than 1 mg/kg.

Doses to representative members of the public from accidental spraying, listed in Table 4-15, exceed 1 mg/kg for the berrypicker for atrazine, bromacil, dalapon, hexazinone, tebuthiuron, and triclopyr. Doses of fosamine to the berrypicker exceed 2 mg/kg. Doses of diuron to the hunter and nearby resident exceed 1 mg/kg; for the berrypicker and fisherman, doses exceed 2 mg/kg. All doses of all other herbicides are less than 1 mg/kg for representative members of the public in spraying accidents.

Doses from Spills

Doses from spill accidents, both direct dermal and via drinking water, are listed in Table 4-16. The spill doses are listed in mg/kg (not micrograms/kg as in the spraying accidents). By far, the highest doses are received in worker spill accidents where workers receive doses exceeding 100 mg/kg for all the herbicides except amitrole, dalapon, picloram, and tebuthiuron. Spills of mixture on workers' skin lead to estimated doses that exceed 10 mg/kg for all herbicides except amitrole, 2,4-DP, and picloram. Doses to the public from truck spills rarely exceed 1 mg/kg. Those from helicopter dumps into a reservoir never exceed 0.01 mg/kg.

Lifetime Doses

Lifetime doses to workers and the public from herbicide spraying for a specified number of exposures over a 70-year lifetime are listed in the final set of tables in Attachment B. Cancer risk based on the specified number of exposures is discussed in Section 5.

Table 4-14

Doses to the Public from Items Receiving the Full per-Acre
Application Rate by Exposure Type,
Accidental-Worst Case
(micrograms/kg)

Herbicide	Direct Dermal	Reentry Hiker	Reentry Picker	Drink Water	Eating Berries	Eating Vegs.	Eating Deer	Eating Bird	Eating Fish
Amitrole	3	0	9	117	93	194	19	116	47
Asulam	209	3	538	73	58	121	13	83	29
Atrazine	355	5	914	125	99	206	22	141	249
Bromacil	417	6	1,075	147	116	242	26	165	59
2,4-D	103	1	264	60	48	99	10	64	24
2,4-DP	133	2	344	73	58	121	13	79	29
Dalapon	417	6	1,075	147	116	242	26	165	59
Dicamba	83	1	215	59	47	97	10	62	23
Diuron	667	10	1,720	235	186	387	42	265	1,878
Fosamine	501	7	1,290	176	140	290	31	199	70
Glyphosate	209	3	538	73	58	121	13	83	29
Hexazinone	250	4	645	88	70	145	16	99	35
Picloram	10	0	26	73	58	121	12	73	29
Simazine	209	3	538	73	58	121	13	83	29
Tebuthiuron	250	4	645	88	70	145	16	99	352
Triclopyr	334	5	860	117	93	194	21	132	47

Table 4-15

Doses for Example People^a for
Accidental-Worst Case Spraying
(micrograms/kg)

Herbicide	Hiker	Berry- picker	Hunter	Fisherman	Nearby Resident
Amitrole	121	222	256	168	314
Asulam	285	878	381	314	406
Atrazine	484	1,492	647	734	690
Bromacil	570	1,755	761	628	812
2,4-D	164	475	239	188	263
2,4-DP	209	609	300	238	330
Dalapon	570	1,755	761	628	812
Dicamba	143	404	215	167	240
Diuron	912	2,809	1,218	2,790	1,299
Fosamine	684	2,107	914	754	974
Glyphosate	285	878	381	314	406
Hexazinone	342	1,053	457	377	487
Picloram	84	167	168	113	204
Simazine	285	878	381	314	406
Tebuthiuron	342	1,053	457	694	487
Triclopyr	456	1,404	609	503	649

^aAll of these people receive multiple exposures as shown in Table 4-5.

Table 4-16

Doses from Herbicide Spills
(mg/kg)

Herbicide	Spill of One Pint Concentrate on Skin	Spill of One Pint Tank Mix on Skin	Helicopter ^a Dump into Pond	Helicopter ^a Dump into Reservoir	Truck ^a Spill into Pond	Truck ^a Spill into Reservoir
Amitrole	1.20	0.24	0.0737	0.0023	0.7365	0.0230
Asulam	240.00	20.04	0.0615	0.0019	0.6150	0.0192
Atrazine	240.00	24.00	0.0737	0.0023	0.7365	0.0230
Bromacil	240.00	12.00	--	--	0.3683	0.0115
2,4-D	144.00	14.40	0.0737	0.0023	0.7365	0.0230
2,4-DP	230.40	9.60	0.0460	0.0014	0.4603	0.0144
Dalapon	--	60.00	0.1841	0.0058	1.8413	0.0575
Dicamba	120.00	12.00	0.0737	0.0023	0.7365	0.0230
Diuron	240.00	19.20	--	--	0.5892	0.0184
Fosamine	240.00	72.00	0.2210	0.0069	2.2095	0.0690
Glyphosate	180.00	30.00	0.0921	0.0029	0.9206	0.0288
Hexazinone	120.00	18.00	0.0552	0.0017	0.5524	0.0173
Picloram	5.76	1.44	0.0921	0.0029	0.9206	0.0288
Simazine	240.00	30.00	0.0921	0.0029	0.9206	0.0288
Tebuthiuron	--	36.00	0.1105	0.0035	1.1048	0.0345
Triclopyr	240.00	48.00	0.1473	0.0046	1.4730	0.0460

^aAssuming 1 liter of water drunk per day.

-- Herbicide not used in a form that could cause this kind of spill.

Appendix D
Human Health Risk
Assessment
(Quantitative)

Section 5

Section 5

HUMAN HEALTH RISK ANALYSIS

This section presents information on potential risks to the health of workers and members of the public from the proposed herbicide applications by comparing the exposure levels estimated in Section 4 with the toxic effect levels described in Section 3. The first subsection describes the methods used to evaluate risks. The second subsection evaluates the risks of threshold effects that include acute toxic effects, chronic systemic effects, and reproductive (fetotoxic and maternal toxic) and teratogenic effects. The last subsection evaluates the risks of the herbicides causing cancer or mutagenic effects in the population at risk. All judgments about risk are discussed in light of the probabilities of the estimated exposures actually occurring.

HOW THE RISKS TO WORKERS AND THE PUBLIC WERE DETERMINED

In this risk analysis, the risks to humans exposed to the 16 herbicides were quantified by comparing the doses estimated in the range of exposure scenarios presented in Section 4 with the results of toxicity tests on laboratory animals described in Section 3. There are two basic approaches for extrapolating from laboratory animal NOEL's to the general human population: the acceptable daily intake approach and the margin-of-safety approach. Under the acceptable daily intake (ADI) approach, "safety factors" based on the quality of the data are applied to either the highest (Thomas 1986; Klaassen and Doull 1980) or lowest (EPA) NOEL dose found in animal studies. These factors have been used for the estimation of acceptable human exposures based on experimental human and animal studies where noncarcinogenic effects were observed following exposure to a toxic chemical substance (Thomas, 1986). An uncertainty factor of 10 has normally been used in the estimation of safe levels in humans from experimental studies when there are valid human studies available and no indication of carcinogenicity. An uncertainty factor of 100 is used when there are few or no human studies available but there are valid long-term animal studies; when there are very limited toxicological data 1,000 or greater could be used to estimate acceptable human exposure.

Safety factors and the "ADI approach" are used by Federal regulatory agencies such as the FDA and EPA to set ADI's for chemicals that a broad segment of the general public are liable to be exposed to for an indeterminate period of time. Thus, the ADI is a lifetime safe dose for threshold toxic effects based on the best available toxicity information on a particular chemical. Cancer and mutation effects are not dealt with in this way since they are not assumed to have a predictable threshold of reversible toxic effects.

The margin-of-safety (MOS) approach used in this risk assessment is based on the same concepts of a threshold of toxicity (approximated by animal no-observed-effect levels (NOEL's) in long-term studies) and of the safety of a dose. However, it differs from the ADI approach in several important ways. First, the MOS approach is not being used here to establish a regulatory standard safe level for the general public against which samples of possibly contaminated products, for example, marketed vegetables or drinking water, would be tested. The margins-of-safety computed here are dose ratios that are direct comparisons of the doses estimated in this risk assessment with the NOEL's from animal studies. For example, an MOS of 100 means the laboratory-determined level is 100 times higher than the estimated dose. Although they correspond with the safety factors used to determine the ADI's, they are applicable only to this risk assessment. It should also be pointed out that a margin-of-safety does not always mean that the dose is safe. A MOS of 3, for example, could represent a high risk of toxic effects for repeated exposures.

Second, the ADI as a standard level for comparison of tested samples should remain relatively stable over the years, modified only when the results of new toxicity tests produce a new NOEL or make a change in the ADI safety factor appropriate. The margins-of-safety in this risk assessment, however, vary with the estimated doses in a particular exposure scenario and are thus used to indicate the potential toxic effects of a proposed chemical under differing conditions or routes of exposure or in comparison with alternative chemicals that may be used for the same purpose.

For doses that are not likely to occur more than once, such as those received by workers spilling a quart of spray mix over their entire upper body, a dose estimate that exceeds the laboratory test animal NOEL does not necessarily lead to the conclusion that there will be toxic effects. All of the NOEL's used in this risk analysis are based on (or take into account) long-term exposure. Estimated doses that exceed the NOEL are compared to the herbicide's acute oral LD₅₀, so that a judgement can be made on the risk of fatal effects. For convenience in this analysis, the ratio between the herbicide's LD₅₀ and the estimated human dose also is expressed as an MOS; however, it should not be interpreted in the same way as the MOS based on a NOEL in terms of the expectation of no effects in humans.

The larger the margin of safety (the smaller the estimated human dose compared to the animal NOEL), the lower the risk to human health. As the estimated dose to humans approaches the animal NOEL (as the MOS approaches one), the risk to humans increases. When an estimated dose exceeds a NOEL (giving an MOS of less than one), the ratio is reversed (the dose is divided by the NOEL) to indicate how high the estimated dose is above the laboratory toxicity level; a minus sign is attached to indicate that the dose exceeded the NOEL; and the result is no longer termed a margin of safety but is called simply a negative ratio.

A ratio of -3, for example, means that the estimated dose is 3 times the laboratory-determined level. A negative ratio infers that the estimated dose (given all the assumptions of the scenario) represents a clear risk of possible acute effects if the ratio is based on the LD₅₀ or of possible chronic effects if the ratio is based on the systemic or reproductive NOEL.

In general when repeated doses to humans approach the animal NOEL (the MOS is less than 10), there is some possibility of harmful effects. In general, when the MOS is less than 100, sensitive individuals may be at risk. Conversely, when the human dose is small compared with the animal NOEL (giving an MOS greater than 100), the risk to humans can be judged negligible. Comparing one-time or once-a-year doses (such as those experienced by the public) to NOEL's derived from lifetime studies tends to greatly overestimate the risk from those rare events.

Systemic effects are evaluated based on the lowest systemic NOEL found in a 2-year feeding study of dogs, rats, or mice. (When subchronic studies reported effects at lower levels than chronic studies, the subchronic NOEL's were used.) Reproductive effects are evaluated based on the lowest maternal, fetotoxic, or teratogenic NOEL found in a three-generation reproductive study or in a teratology study.

A worst case analysis of cancer risk is conducted for the herbicides for which there are positive cancer studies, amitrole, asulam, atrazine, bromacil, picloram, 2,4-D and 2,4-DP, and for herbicides for which there is scientific controversy about their ability to cause cancer, such as and glyphosate. The risk of cancer is calculated for an individual by comparing estimates of lifetime dose over a 70-year period (computed in Section 4) with cancer potency estimates derived in the Hazard Analysis section. A worst case analysis is also conducted for those herbicides that have positive mutagenicity tests or those for which no data is available. The risk of these herbicides causing mutations is qualitative rather than quantitative, with a statement of the probable risk based on the available evidence of mutagenicity and carcinogenicity.

RISK OF GENERAL SYSTEMIC AND REPRODUCTIVE EFFECTS

For each application scenario, routine-realistic, routine-worst case, and accidental worst case assumptions were used to compute margins of safety for workers and the public for all 16 herbicides. Complete tables for each herbicide are given in Attachment C. The margins of safety were computed by comparing the laboratory-determined NOEL's and LD₅₀'s in Table 5-1 with the doses shown in Attachment B.

Risk to the Public Under Routine Operations

Table 5-2 summarizes the margin-of-safety results for the public for the 16 herbicides under both the routine-realistic and routine-worst case exposure scenarios.

Risk to the Public Under Routine-Realistic Scenarios

Margins of Safety for the Public Under Routine-Realistic Scenarios. Table 5-2 shows that there are large margins of safety (greater than 150) for every category of exposure--even cumulative exposures--under the routine-

Table 5-1

Toxicity and Cancer Potency of Herbicides

Herbicide	Rat LD ₅₀ mg/kg	Systemic NOEL mg/kg/day	Reproductive/ Terat. NOEL mg/kg/day	Cancer Potency per mg/kg/day
Amitrole	4,080.00	0.025	5.0	1.4
Asulam	4,000.00	50.0	50.0	0.02
Atrazine	1,869.00	3.7	100.0	0.03
Bromacil	3,998.00	6.25	7.92	0.0038
2,4-D	375.00	1.0	5.0	0.0050
2,4-DP	532.00	5.0	6.25	0.059
Dalapon	7,577.00	15.0	300.0	*
Dicamba	757.00	25.0	2.5	*
Diuron	3,750.00	0.625	6.25	*
Fosamine	24,400.00	25.0	500.0	*
Glyphosate	4,320.00	31.0	10.0	0.000024
Hexazinone	1,690.00	10.0	125.0	*
Picloram	8,200.00	7.0	50.0	0.00057
Simazine	5,000.00	5.0	5.0	*
Tebuthiuron	644.00	12.5	90.0	*
Triclopyr	630.00	2.5	10.0	*

*No oncogenic potential was indicated from laboratory studies, therefore a cancer potency analysis was not conducted.

realistic scenario for asulam, atrazine, bromacil, 2,4-D, 2,4-DP, dalapon, dicamba, diuron, fosamine, glyphosate, hexazinone, picloram, simazine, and tebuthiuron, and triclopyr. Although the public should not be chronically exposed to these herbicides (indeed, given the remote location of most spray areas, it is unlikely that any member of the public will be exposed at all), these large margins of safety mean that they could be repeatedly exposed to these levels, or cumulatively exposed to these levels, and suffer no adverse effects. This is true for all individuals including pregnant women and the vast majority of sensitive individuals.

(insert Table 5-2)

Lowest Margins of Safety for the General Public Under the Routine Scenarios

Herbicide	Routine-Worst Case ^a Scenarios	
	Routine-Realistic Scenarios	
Amitrole	All right-of-way and most backpack MOS's are greater than 50, except eating vegetables (41), and residents (32). Most MOS's for aerial application are less than 50, including the following that are less than 10: berry-picker (8), hunter (9), fisherman (9), and residents (6).	Many of the right-of-way and 2 of the backpack MOS's are less than 50. Most of the aerial MOS's are less than 50, including the following that are less than 10: drinking water (2), eating berries (2), eating vegetables (1), eating bird (4), eating fish (4), hiker (1), berry-picker (1), hunter (1), fisherman (1), and resident (-1).
Asulam	All situations 8,000 or greater.	All situations greater than 860.
Atrazine	All situations greater than 310.	All situations greater than 50 except for berry-pickers (44), for the 400-acre aerial application.
Bromacil	All situations are greater than 2,200.	All situations are 700 or greater.
2,4-D	All situations greater than 150.	All right-of-way and backpack MOS's are greater than 380. All aerial MOS's are greater than 10 with the following situations having MOS's of 10 to 50: vegetation contact by picker (38), eating vegetables (48), hiker (43), berry-picker (17), hunter (32), fisherman (36) and resident (23).
2,4-DP	All situations 990 or greater.	All situations greater than 120.
Dalapon	Greater than 1,100 in all situations.	All situations greater than 100 except berry-picker (72).

^aA margin of safety of 50 was chosen as the cutoff to report values in this table for ease of comparison. It was not intended to indicate what would be considered low or high risk.

Table 5-2 (Cont.)

Herbicide	Routine-Realistic Scenarios	Routine-Worst Case ^a Scenarios
Dicamba	Greater than 1000 in all situations.	All situations greater than 100 except for berrypickers (47), hunters (85), fishermen (95), and residents (60) for 400-acre aerial application.
Diuron	Greater than 180 in all situations.	All MOS's 77 or greater in all situations.
Fosamine	All greater than 2,600.	All 99 or greater.
Glyphosate	All greater than 1,500.	All 95 or greater.
Hexazinone	All greater than 1,200.	All greater than 150.
Picloram	All greater than 3,400.	All greater than 150.
Simazine	All greater than 390.	All greater than 50 except for berrypickers (48) in 400-acre aerial application.
Tebuthiuron	All greater than 2,500.	All MOS's 99 or greater.
Triclopyr	All greater than 390.	All situations greater than 50 except for vegetation contact by berrypicker (29), and the multiple routes for hiker (42), berrypicker(15), hunter (32), fisherman (36), and resident (25) in the 400-acre aerial application.

^aA margin of safety of 50 was chosen as the cutoff to report values in this table for ease of comparison. It was not intended to indicate what would be considered low or high risk.

Amitrole has many situations where the margin of safety is less than 20 for systemic effects. (The margin of safety for reproductive effects is greater than 1,000--indicating negligible risk for reproductive effects.) The greatest risk under the aerial application scenario is for individuals who drink a liter of water from a shallow stream 50 feet from the treatment area immediately after application, or eat vegetables from within 600 feet of the treatment area immediately after application. For this reason, all of the amitrole cumulative exposures for the berrypicker, hunter, fisherman, and residents are less than 10. This indicates that people chronically exposed to these levels of amitrole could experience thyroid problems. The large ratios compared to the LD₅₀ indicate very little chance of acute effects.

The greatest risk by exposure route occurs in contacting vegetation that has just been sprayed with one of the herbicides while picking berries and in eating vegetables that have received spray drift. Because of this, the representative members of the public at greatest risk from any of the herbicides are the nearby resident and the berrypicker. Exposure routes leading to least risk are direct dermal exposure to spray drift, drinking water with drift residues, and eating animals or fish that have drift residues. Persons at least risk are the hunter, hiker, and fisherman. These relationships hold for all 16 herbicides.

MOS's for all herbicides estimated in the three routine-realistic public exposure scenarios are given in Attachment C. MOS's for the three most heavily used herbicides, 2,4-D, glyphosate, and triclopyr begin in Tables C-57, C-93, and C-123, respectively.

Probability of Estimated Routine-Realistic Public Doses Occurring. Although these three scenarios represent what can happen under routine operations, the probability of people receiving doses as high as those projected here is quite low.

There are no residents, hikers, fishermen, or berrypickers in the vicinity of the vast majority of treatment units. Additional precautions, such as posting the area, are normally used to ensure that no one would be exposed during or immediately after a herbicide application operation.

Moreover, as described in Section 4, these routine-realistic scenarios use a number of conservative assumptions that tend to overestimate rather than underestimate what is expected in the majority of operations. For example, predicted levels in water (which determine doses for drinking water, eating fish, and all of the cumulative exposures) are 100 times higher than levels seen in extensive field testing. Extensive monitoring studies conducted by the Forest Service for phenoxy herbicides from 1974 to 1978 showed negligible levels of herbicides in streams (all were less than 0.04 parts per million). These extremely low levels were found despite the fact that during the 1974-78 period not all herbicide applications were monitored. Only those applications most likely to result in significant residues or cause for public concern were actually monitored (USDA, 1980).

The levels predicted on berries are also higher than those found in similar forest plants (USDA, 1984). In addition, the levels predicted for deer in the routine-realistic scenario are similar to the highest levels found by Newton and Norris (1968, as cited in Dost, 1983), who did not find levels greater than 0.08 parts per million in edible deer tissues.

Risk to the Public Under Routine-Worst Case Scenarios

The routine-worst case scenarios described in Section 4 were intended to indicate the upper bound for public exposure to herbicide applications in the Pacific Northwest. The low probability of each assumption, which would apply to all of the events that led to the exposures described in Table 5-2, must be emphasized. It is extremely unlikely that anyone would receive a dose as high as those estimated here.

Margins of Safety Under Routine-Worst Case Scenarios. Table 5-2 indicates that most margins of safety projected under this routine-worst case scenario are greater than 50 except for amitrole, 2,4-D, and triclopyr. Under these extreme assumptions, risk to individuals is very low except for people who receive multiple exposures from a 400-acre fixed wing application. Amitrole has a number of situations where the margin of safety is less than 10. For amitrole, people repeatedly receiving doses as high as predicted here over a long period could experience thyroid problems. Margins of safety calculated for combined routes of exposure to 2,4-D and triclopyr were all within 10 to 50 in the worst case aerial application. Chronic doses of 2,4-D, as predicted by this analysis, could affect the peripheral nervous system which, in most cases, would be a reversible effect. For triclopyr, people who chronically receive doses predicted here could experience kidney problems. Because the margins of safety were computed by comparing acute exposures with chronic no-effect levels, the risk of occurrence of these effects can be considered extremely low, especially considering the extreme unlikelihood of nearby residents receiving repeated doses over the long term. The margin of safety derived for triclopyr also is extremely conservative because the toxic effects observed in dogs that resulted in a systemic NOEL of 2.5 mg/kg/day may have been exacerbated by the decreased renal excretion capacity of dogs, which is not representative of human renal physiology. Feeding studies in other test species did not result in kidney problems or other toxic effects at a higher dose level (30 mg/kg/day) (USDA, 1984).

The margins of safety computed for the residents in the worst case 400-acre aerial spray site indicate that sensitive individuals could suffer some acute toxic effects from the predicted exposures to amitrole, atrazine, 2,4-D, dicamba, simazine, and triclopyr. For atrazine, all margins of safety are greater than 50 except for a berry picker who is dermally exposed for 4 hours, eats 0.9 pounds of berries and drinks a liter of water contaminated at the highest possible level. This person, as well as the people who receive multiple exposures to dicamba and simazine listed in Table 5-2, could experience some systemic effects. However, that is unlikely because these are one-time doses and are all more than 10,000 times lower than the LD₅₀. It should be noted that the systemic NOEL for atrazine is based on weight loss in dogs. Therefore, sensitive individuals could become ill and possibly experience stomach problems. For simazine, no toxic effects were seen at the highest dose tested in two chronic exposure studies. Therefore, any toxic effects resulting from these routine-worst case doses should be limited if any.

Public MOS's for all 16 herbicides are presented in Attachment C. MOS's for the doses estimated in the routine-worst case public exposure scenarios for the three most heavily used herbicides--2,4-D, glyphosate, and triclopyr--begin in Tables C-60, C-96, and C-126, respectively.

Probability of the Routine-Worst Case Doses Occurring. The probability of someone receiving a dose as high as those predicted under the routine-worst case scenario is negligible. The probability is low because this scenario assumes that a number of unlikely events occur simultaneously. For example, using the assumptions that the probability of treating a unit as big as 400 acres is 1 in 100, and the probability of the high drift case is 1 in 100, and the probability of someone being in the vicinity of the treatment area is 1 in 100, then the probability of someone receiving a dose as high as those predicted here is 1 in 1 million ($0.01 \times 0.10 \times 0.01 = 0.000001$). In fact, historical records indicate that the probability of these events occurring simultaneously is less than this.

Risk to the Public Under Accidental Scenarios

Table 5-3 summarizes the risk to the public from direct exposure to aerial applications or from eating food or drinking water that has been directly hit at the highest application rate. The relatively low margins of safety for amitrole, atrazine, bromacil, 2,4-D, diuron, and triclopyr indicate that people exposed to a direct aerial application or exposed to items that received the highest application rate could experience some toxic effects. The extent of effects would depend upon their duration of exposure and any precautionary measures that were taken. For example, if people gathered a bushel of berries from a spray area and did not wash them but froze them and then ate them every day for a month, they might feel quite ill. However, if people bathed after being in the forest or washed food items before eating them, the doses would drop (and thus increase the margins of safety) substantially.

The risk of a member of the public being directly hit by an aerial spray operation is very small. The probability of a pesticide application in an area not scheduled for treatment is low. According to the Forest Service data on insecticide application (USDA, 1984), the probability, based on empirical data, of some kind of significant error in a pesticide application is 0.0002 (at the 95-percent confidence level). Operational features of herbicide operations make the probability of applying a herbicide in an area not scheduled for treatment less than that of insecticide operations. Using this value as an extremely conservative estimate of the probability of an application directly hitting a human being, there might be three accidents over a period of 8 years if a spraying operation occurred every day for 6 months during each of those years. In addition, the probability that someone will be in the area being sprayed is very low because normally the area is posted before spraying and humans will be kept out of the treated areas during spray operations. Thus, the probability of such accidents can be considered negligible.

Table 5-3

Margins of Safety Less Than 10 for the General Public
in the Accidental-Worst Case Scenarios

Herbicide	MOS Less Than 10	
	Items Sprayed at Full Application Rate	Spill
Amitrole	<p>All exposures except vegetation contact by the hiker are less than 10.</p> <p>Doses from all other routes except eating deer meat exceed the NOEL.</p>	Doses from helicopter and truck spill into pond and from truck spill into reservoir exceed the NOEL.
Asulam	None less than 10 for either scenario.	
Atrazine	Vegetation contact by berry picker; combined exposure routes for hiker, berry picker, hunter, fisherman, and resident.	Truck spill into pond.
Bromacil	Vegetation contact by the berry picker, combined exposures for berry picker, hunter, fisherman, and resident.	Truck spill into pond.
2,4-D	Vegetation contact by berry picker, combined exposure routes for hiker, berry picker, hunter, fisherman, and resident.	Truck spill into pond dose exceeds NOEL.
2,4-DP	Only MOS for combined exposure of the less than 10.	Truck spill into pond.

Table 5-3 (Cont.)

Herbicide	MOS Less Than 10	
	Items Sprayed at Full Application Rate	Spill
Dalapon	Only the combined exposure berrypicker MOS is less than 10.	Truck spill into pond
Dicamba	Combined exposure of the berrypicker.	Truck spill into pond.
Diuron	All margins of safety, except hiker vegetation contact and person eating deer are less than 10. The berrypicker vegetation contact and eating fish exposures and all combined exposures exceed the systemic NOEL.	Truck spill into pond dose exceeds NOEL
Fosamine	None less than 10.	Truck spill into pond.
Glyphosate	None less than 10.	Truck spill into pond.
Hexazinone	Only the combined exposure berrypicker MOS is less than 10.	Truck spill into pond.
Picloram	None less than 10.	Truck spill into pond.
Simazine	Berrypicker vegetation contact and combined routes for berrypicker.	Truck spill into pond.
Tebuthiuron	None less than 10.	Truck spill into pond.

Table 5-3 (Cont.)

Herbicide	MOS Less Than 10	
	Items Sprayed at Full Application Rate	Spill
Triclopyr	Vegetation contact by the berrypicker; combination exposure for hiker, hunter, fisherman, and resident.	Truck spill into pond dose exceeds NOEL.

Again it must be noted that these are one-time, rather than repeat or chronic, exposures and that the comparison of these doses with the acute LD₅₀'s shows that no one is at risk of fatal effects. Complete margins of safety computed for each chemical and application under the accidental worst-case scenario are presented in Attachment C.

Risk to the Public from Herbicides Used in Brown and Burn Operations

Brown and burn operations are conducted on approximately 500 to 1,500 acres of Forest Service and BLM land every year. These operations are generally limited to the coast range of Oregon on brushy hardwood vegetation and often steep terrain. 2,4-D accounts for approximately 75 percent of the total herbicide used during this type of operation. Glyphosate and triclopyr are used to a much lesser extent. Typically, the selected herbicide is applied aerially in the fall and the vegetation is not burned until the following spring, approximately 5 to 7 months later. However, in some cases burning may take place as soon as 2 weeks after the herbicide has been applied. The treatment units average approximately 30 acres. Crew size at any given site may vary from 10 to 26 workers during the burning operation.

To estimate worker exposure during these operations, it is necessary to calculate the amount of herbicide that will be remaining on the vegetation at the time of burning. The half-lives for 2,4-D, glyphosate, and triclopyr are 16, 14, and 18 days, respectively. Therefore, after 2 weeks, residues of the amount of applied herbicides would remain on the vegetation: 54 percent of 2,4-D, 50 percent of glyphosate, and 58 percent of triclopyr.

The following assumptions were used to calculate potential worker exposure to smoke:

1. 32 metric tons (32,000 kg) of fuel per acre
2. 40 percent of the available fuel by weight is burned
3. Smoke density is 5 mg/m³ (visibility 100 m)
4. 8.5 g smoke/kg of fuel burned
5. 30 acres per average treatment unit
6. All herbicide residue is released to the atmosphere upon burning
7. Respiration rate for workers at moderate work is 29 liters per minute or 1.74 m³ per hour

$32,000 \text{ kg/acre} * 0.40 = 13,000 \text{ kg of fuel per acre}$

$13,000 \text{ kg} * 8.5 \text{ g/kg} = 110,000 \text{ g of smoke produced per acre}$

$110,000 \text{ g} / (5 \text{ mg/m}^3) = 22,000,000 \text{ m}^3 \text{ of smoke per acre}$

$22,000,000 \text{ m}^3 * 30 \text{ acres} = 6.6 \times 10^8 \text{ m}^3$ of smoke per treatment unit

2,4-D. The number of kilograms of 2,4-D applied at 5.7 lb/acre is: $5.7 \text{ lb} / (2.2 \text{ lb/kg}) = 2.6 \text{ kg}$

The amount of 2,4-D available after 2 weeks is: $2.6 \text{ kg} * .54 = 1.4 \text{ kg}$ of 2,4-D per acre.

The atmospheric concentration of 2,4-D is: $1.4 \times 10^6 \text{ mg} / 2.2 \times 10^7 \text{ m}^3 = .0636 \text{ mg/m}^3$

The expected dose of 2,4-D for an average worker (50 kg body weight) respiring at a rate of $1.74 \text{ m}^3 / \text{hour}$ is: $(.0636 \text{ mg/m}^3 * 1.74 \text{ m}^3 / \text{hour}) / 50 \text{ kg} = 2.2 \times 10^{-3} \text{ mg/kg/hour}$.

This is far below the NOEL of 1.0 mg/kg/day. A 1-hour-per-day exposure to smoke of this density is a reasonable expectation. If a worker were exposed to 3 hours of smoke per day, the dose would be only 3 times greater than that calculated and would still be well below the NOEL. A worker is not typically exposed to brown and burn operations more than 12 days per year. Therefore, the overall health risk from this type of operation with 2,4-D is negligible.

Glyphosate. The number of kilograms of glyphosate applied at 5.0 lb/acre is:

$5.0 \text{ lb} / (2.2 \text{ lb/kg}) = 2.3 \text{ kg}$

The amount of glyphosate available after 2 weeks is: $2.3 \text{ kg} * .50 = 1.15$ of glyphosate per acre.

The atmospheric concentration of glyphosate is: $1.15 \times 10^6 \text{ mg} / 2.2 \times 10^7 \text{ m}^3 = .0523 \text{ mg/m}^3$.

The expected dose of glyphosate for an average worker (50 kg body weight) respiring at a rate of $1.74 \text{ m}^3/\text{hour}$ is: $(.0523 \text{ mg}/\text{m}^3 * 1.74 \text{ m}^3/\text{hour}) / 50 \text{ kg} = 1.82 \times 10^{-3} \text{ mg}/\text{kg}/\text{hour}$. This dose is well below the NOEL of greater than $31 \text{ mg}/\text{kg}/\text{day}$ and should pose no risk to health.

Triclopyr. The number of kilograms of triclopyr at a maximum application rate of $8.0 \text{ lb}/\text{acre}$ is: $8.0 / (2.2 \text{ lb}/\text{kg}) = 3.6 \text{ kg}$

The amount of triclopyr available after 2 weeks is: $3.6 \text{ kg} * .58 = 2.09 \text{ kg}$ of triclopyr.

The atmospheric concentration of triclopyr is: $2.09 \times 10^6 \text{ mg} / 2.2 \times 10^7 \text{ m}^3 = .095 \text{ mg}/\text{m}^3$

The expected dose of triclopyr for an average worker (50 kg body weight) respiring at a rate of $1.74 \text{ m}^3/\text{hour}$ is: $(.095 \text{ mg}/\text{m}^3 * 1.74 \text{ m}^3/\text{hour}) / 50 \text{ kg} = 3.31 \times 10^{-3} \text{ mg}/\text{kg}/\text{hour}$. This dose is insignificant and is far below the NOEL of $2.5 \text{ mg}/\text{kg}/\text{day}$. No adverse health effects are expected.

Combustion Project. Wall et al. (1984) studied the pyrolysis products of 2,4-D and glyphosate. At 800°C 2,4-D volatilized and condensed upon cooling. Approximately 2.5 percent was converted to 2,4-dichlorophenol. No other significant pyrolysis products were formed.

Temperatures during the burning of vegetation are expected to reach at least 1,000 degrees Celsius, which could cause considerable breakdown of the herbicides (Kennedy et al., 1969). Glyphosate was almost completely destroyed at 800 degrees and largely converted to CO_2 . Approximately 5 to 6 percent of the glyphosate was converted to volatile by-products, including primarily acetonitrile, alkylpyridines, and alkylpyrazines. To a lesser extent benzene, toluene, and acetic acid were formed. No information on the combustion of triclopyr is available.

Cancer Risk from Burning Vegetation

Dost (1986) has performed a cancer risk assessment of the main carcinogens found in wood smoke, the polynuclear aromatic hydrocarbons (PAH's). This chemical group includes benzo(a)pyrene (BaP), a known human carcinogen with a cancer potency of .0033. Other PAH's in wood smoke which have shown to be carcinogenic in laboratory animal studies include benzo(c)phenanthrene, benzo(a)fluoranthrene, 3-methyl-cholanthrene, and dimethylbenzanthrene. These all have potencies less than or equal to BaP.

Dost used the following assumption in estimating BaP exposure:

- o 2,500 mg BaP is released from every kg of fuel that is burned (EPA finds this number sufficiently conservative).
- o All BaP is incorporated into fine particulate matter that is of respirable size and 8,500 mg of particulate is produced per kg of fuel that is burned (the Forest Service frequently employs this number).
- o Smoke density is 0.155 mg/m^3 based on a visibility of 2 miles.
- o A person is exposed for 6 hours per day, 20 days per year, for 10 years of his lifetime.

Based on these assumptions, the average lifetime exposure concentration would be $9.1 \times 10^{-5} \text{ ug/m}^3$ of BaP. The increased cancer risk due to BaP would be 3×10^{-7} or 3 in 10,000,000.

Dost completed similar calculations for the four other carcinogenic PAH's. The total increased risk of cancer from the PAH's, including BaP, is 1.1×10^{-6} .

Therefore, based on these assumptions there is very little risk of adverse health effect from exposure to weed smoke from BLM burning operations.

Risk to Workers from Routine Operations

Tables 5-4 to 5-7 summarize the margins of safety for workers based on the systemic and reproductive NOEL's for the 16 herbicides. Full tables showing margins of safety computed for the 16 herbicides are presented in Attachment C, Tables C-1 through C-160. Because of the assumptions that were made to overestimate risk, the Forest Service and BLM estimate that almost all of the operations that take place will fall within the values predicted for the routine-realistic scenarios. Routine-worst case estimates are presented to show the upper bound or 95-percent confidence level of risk to workers.

It must be emphasized that the routine worker exposures and resultant margins of safety are what could be expected in the majority of vegetation management programs in the Pacific Northwest for workers not wearing protective clothing or equipment. All of the studies from which the routine-realistic exposures were calculated are based on workers wearing no protective clothing. The use of protective clothing can substantially reduce worker doses, as shown in field studies of worker exposure, and thereby increase their margins of safety.

Effects of the Use of Protective Clothing

Protective clothing can reduce worker exposures by 27 to 99 percent as shown in a number of relevant field studies. The calculated doses presented below were based on the assumption that workers work with bare hands and wear ordinary work clothing, such as cotton pants and short-sleeve shirts. It is common practice, however, for herbicide applicators to wear clothing that affords more protection. Typical clothing often includes long-sleeve shirts or coveralls, gloves and hats.

Research has shown that such protective clothing can substantially reduce worker exposure. For example, in right-of-way spraying, doses of spray gun applicators wearing clean coveralls and gloves were reduced by 68 percent compared to the doses they got without this protection (Libich et al., 1984). During an aerial spraying operation, mixer/loaders wearing protective clothing reduced their exposure by 27 percent and other crew members reduced their exposure by 58 percent compared to the levels observed without precautions (Lavy et al., 1982).

During insecticide applications to orchards, mixers reduced their exposure by 35 percent and sprayers reduced their exposure by 49 percent by wearing coveralls (Davies et al., 1982). Putnam and coworkers found that nitrofen applicators and mixer/loaders wearing protective clothing reduced their exposure by 94 to 99 percent compared to the doses experienced without protection (Waldron, 1985). Although protective clothing generally does reduce worker exposure and resulting doses, the degree of protection depends on the application system, the work practices, and the specific herbicide. In one extreme case, workers wearing protective clothing did not receive significantly lower doses than workers with less clothing (Lavy et al., 1984). In this case backpack applicators had to treat and move through dense vegetation that was taller than themselves.

Most exposure to herbicide applicators is dermal, not inhalation (Kolmodin-Hedman, et al., 1983), so the use of respirators is ineffective and unnecessary. The hands are the site of the greatest potential herbicide exposure, and rubber gloves are generally quite effective in preventing exposure to hands (Putnam et al., 1983).

Based on the review of field studies protective clothing was normally found to reduce worker doses by the following amounts:

<u>Type of Worker</u>	<u>Percent Reduction in Dose</u>
1. Right-of-way applicators	68.1
2. Aerial application crew members	57.1
3. Aerial mixer/loaders	27.1
4. Injection bar applicators	54.7
5. Hack-and-squirt applicators	57.6

Doses to protected backpack applicators were based on doses to right-of-way applicator who used hand-held nozzles. Tables 5-4 and 5-5 list routine-realistic margins of safety computed for workers without protective clothing and for workers with protective clothing in parentheses. Tables 5-6 and 5-7 list the same values for routine-worst case doses.

Risk to Workers Under Routine-Realistic Scenarios

In the routine-realistic scenario, all categories of workers applying asulam or picloram have MOS's greater than 100. This indicates that even workers chronically exposed to these herbicides should suffer no ill effects. For all the other herbicides, as shown in Tables 5-4 and 5-5, at least one category of worker (primarily backpack sprayers) had MOS's less than 100 in the routine-realistic scenario. This means that unprotected sensitive workers that routinely receive doses this high may experience some toxic effects from applying these herbicides in certain situations.

Backpack sprayers are clearly at greatest risk based on comparisons of estimated doses with systemic and reproductive NOEL's for all of the herbicides. Hand applicators are next, while pilots and mixer-loaders are at somewhat lower risk. Observers and right-of-way applicators are at least risk.

Except for backpack sprayers using asulam or picloram, all backpack sprayers have margins of safety less than 100. Amitrole, atrazine, bromacil, 2,4-D, and triclopyr have MOS's less than 10; and in the case of diuron, the dose exceeds the NOEL. The doses and margins of safety are based on 6 hours per day of exposure. Any reduction in the time of exposure would reduce the dose and increase the margin of safety proportionally.

Diuron appears to be the herbicide presenting the greatest risk from repeated exposures. Backpack sprayers using diuron in the routine-realistic scenario receive a dose that is less than the systemic NOEL. Diuron systemic MOS's for hack-and-squirt applicators are also less than 10 in the routine-realistic case.

2,4-D presents the next highest long-term risk. Backpack sprayers using 2,4-D in the routine-realistic scenarios receive doses that have systemic margins of safety less than 10. Pilots, mixer/loaders, and both types of hand applicators have MOS's less than 50.

Amitrole and triclopyr have at least four margins of safety for workers (including those for backpack sprayers) that are less than 100.

Risk to Workers Under Routine-Worst Case

As shown in Tables 5-6 and 5-7, a number of herbicides have margins of safety less than 10 in the routine-worst case scenario.

Backpack sprayers using diuron, triclopyr, 2,4-D, or amitrole in the routine-worst case scenario receive doses that exceed their respective systemic NOEL's. In addition, doses calculated for truck applicators and hack and squirt applicators using diuron exceed the systemic NOEL. Margins of safety for the reproductive NOEL's are much higher. No applicator dose exceeds the reproductive NOEL for any herbicide in any situation.

All categories of workers, except the aerial supervisor and observer, have margins of safety less than 10 for at least one of the herbicides. Picloram and asulam are the only herbicides that have margins of safety greater than 20 for all categories of workers.

The probability of workers receiving repeated daily doses as high as predicted here is extremely low (less than 1 chance in 1,000). Therefore, even if a worker were to feel ill for a day or so from an unusually high dose, he would not be expected to suffer permanent damage. The vast majority of the time workers will be receiving doses less than those predicted in the routine-realistic scenario. Sensitive individuals would be at greater risk.

The routine-worst case analysis for workers is based on a series of assumptions that, acting together, greatly increase the estimated risk. It uses the upper 2.5 percent of doses received in field studies, the highest application rates used by BLM and the Forest Service, and the longest work hours for each type of project.

If we combine the probability of getting a dose above the upper 95 percent confidence level of field studies (1 in 40), with a probability of using the highest application rate of 1 in 20, and a probability of someone working the maximum hours of 1 in 20, then the probability of a worker getting a dose as high as predicted here is 1 in 16,000. ($1/40 \times 1/20 \times 1/20 = 1/16,000$).

Risk to Workers from Spilling Concentrate or Spray Mix on Their Skin

It must be noted at the outset when considering the effects resulting from these types of accidents that the doses estimated here are based on dermal penetration levels derived in studies over many days; these chemicals do not penetrate the skin immediately but over a considerable period of time.

Table 5-4

Worker Margins of Safety for Reproductive Effects
Routine-Realistic Scenarios

Herbicide	Aerial			Back Pack		Truck	Hand Application		
	PILOT	MIX/L	SUP	OBS	BP		APPL MIX/L	H&S	INJ BAR
Amitrole	++(++)	++(++)	++(++)	++(++)	++(++)	++(++)	++(++)	++(++)	++(++)
Asulam	++(++)	720(990)	++(++)	++(++)	250(810)	++(++)	++(++)	--	--
Atrazine	++(++)	920(++)	++(++)	++(++)	200(650)	++(++)	++(++)	--	--
Bromacil	--	--	--	--	12(38)	580(++)	410(790)	71(170)	190(410)
2,4-D	170(++)	120(160)	++(++)	++(++)	26(82)	++(++)	++(++)	74(180)	200(++)
2,4-DP	240(580)	170(230)	++(++)	++(++)	30(95)	++(++)	810(++)	58(140)	150(340)
Dalapon	++(++)	++(++)	++(++)	++(++)	450(++)	++(++)	++(++)	--	--
Dicamba	250(590)	170(240)	++(++)	++(++)	61(190)	++(++)	++(++)	45(110)	120(260)
Diuron	--	--	--	--	9.5(30)	460(++)	320(620)	56(130)	150(330)
Fosamine	++(++)	++(++)	++(++)	++(++)	++(++)	++(++)	++(++)	++(++)	++(++)
Glyphosate	250(590)	170(240)	++(++)	++(++)	40(130)	++(++)	++(++)	--	--
Hexazinone	++(++)	++(++)	++(++)	++(++)	680(++)	++(++)	++(++)	--	--
Picloram	++(++)	++(++)	++(++)	++(++)	++(++)	++(++)	++(++)	++(++)	++(++)
Simazine	62(150)	43(59)	410(970)	++(++)	15(49)	730(++)	520(++)	--	--
Tebuthiuron	++(++)	++(++)	++(++)	++(++)	360(++)	++(++)	++(++)	--	--
Triclopyr	250(590)	170(240)	++(++)	++(++)	30(97)	++(++)	++(++)	90(210)	240(520)

Numbers in parentheses refer to margins of safety for workers wearing protective clothing.

MIX/L = Mixer/Loader

SUP = Supervisor

OBS = Observer

BP = Backpack

APPL = Sprayer

H&S = Hack and Squirt

INJ BAR = Injection Bar

++ = MOS of 1000 or more

-- = Not used in respective operation

Table 5-5
Worker Margins of Safety for Systemic Effects
Routine-Realistic Exposures

Herbicide	Aerial			Back Pack	Truck			Hand Application		
	PILOT	MIX/L	SUP		OBS	BP	APPL	MIX/L	APPL MIX/L	H&S
Amitrole	62(150)	43(59)	410(970)	++(++)	7.6(24)	360(++)	360(490)	260(500)	45(110)	120(260)
Asulam	++(++)	870(990)	++(++)	++(++)	300(810)	++(++)	++(++)	++(++)	--	--
Atrazine	49(120)	34(47)	320(760)	++(++)	7.5(24)	360(++)	350(480)	260(490)	--	--
Bromacil	--	--	--	--	9.5(30)	460(++)	450(610)	320(620)	56(130)	150(330)
2,4-D	33(79)	23(32)	220(520)	++(++)	5.1(16)	190(620)	190(260)	140(270)	15(35)	39(87)
2,4-DP	190(460)	140(190)	++(++)	++(++)	24(76)	910(++)	890(++)	650(++)	47(110)	120(270)
Dalapon	190(440)	130(180)	++(++)	++(++)	23(73)	++(++)	++(++)	780(++)	--	--
Dicamba	++(++)	++(++)	++(++)	++(++)	610(++)	++(++)	++(++)	++(++)	450(++)	++(++)
Diuron	--	--	--	--	-1.1(3.0)	46(150)	45(61)	32(62)	5.6(13)	15(33)
Fosamine	410(990)	290(400)	++(++)	++(++)	51(160)	++(++)	++(++)	++(++)	220(530)	590(++)
Glyphosate	770(++)	540(740)	++(++)	++(++)	130(410)	++(++)	++(++)	++(++)	--	--
Hexazinone	200(470)	140(190)	++(++)	++(++)	54(170)	++(++)	++(++)	830(++)	--	--
Picloram	++(++)	++(++)	++(++)	++(++)	880(++)	++(++)	++(++)	++(++)	++(++)	++(++)
Simazine	62(150)	43(59)	410(970)	++(++)	15(49)	730(++)	720(980)	520(++)	--	--
Tebuthiuron	620(++)	430(590)	++(++)	++(++)	51(160)	++(++)	++(++)	++(++)	--	--
Triclopyr	62(150)	43(59)	410(970)	++(++)	7.6(24)	360(++)	360(490)	260(500)	22(53)	59(130)

Numbers in parentheses refer to margins of safety for workers wearing protective clothing.

MIX/L = Mixer/Loader
SUP = Supervisor
OBS = Observer
BP = Backpack
APPL = Sprayer
H&S = Hack and Squirt
INJ BAR = Injection Bar
++ = MOS of 1000 or more
-- = Not used in respective operation

Table 5-6

**Worker Margins of Safety for Reproductive Effects
Routine-Worst Case Scenarios**

Herbicide	Aerial			Back Pack		Truck			Hand Application		
	PILOT	MIX/L	SUP	OBS	BP	APPL	MIX/L	APPL MIX/L	H&S	INJ BAR	
Amitrole	750(++)	590(800)	++(++)	++(++)	160(520)	++(++)	++(++)	++(++)	790(++)	++(++)	
Asulam	89(210)	70(96)	520(++)	++(++)	24(77)	190(600)	330(450)	280(540)	--	--	
Atrazine	150(360)	120(160)	860(++)	++(++)	40(130)	220(710)	380(530)	330(630)	--	--	
Bromacil	--	--	--	--	1.3(4.1)	15(48)	26(36)	22(43)	6.3(15)	23(52)	
2,4-D	12(30)	9.8(13)	72(172)	++(++)	3.4(11)	38(120)	66(92)	56(110)	6.6(16)	24(54)	
2,4-DP	23(56)	18(25)	130(320)	760(++)	3.7(12)	37(120)	64(88)	55(100)	5.2(12)	19(43)	
Dalapon	180(430)	140(190)	++(++)	++(++)	40(130)	570(++)	980(++)	840(++)	--	--	
Dicamba	7.5(18)	5.9(8.0)	43(100)	240(580)	2.0(6.5)	26(84)	45(62)	39(75)	4.0(9.3)	15(33)	
Diuron	--	--	--	--	1.7(5.4)	7.4(24)	13(18)	11(21)	5.0(12)	19(41)	
Fosamine	250(590)	190(270)	++(++)	++(++)	73(230)	++(++)	++(++)	++(++)	390(++)	++(++)	
Glyphosate	12(29)	9.4(13)	69(160)	390(920)	3.2(10)	38(120)	65(90)	56(110)	--	--	
Hexazinone	250(590)	200(270)	++(++)	++(++)	67(220)	390(++)	680(930)	580(++)	--	--	
Picloram	++(++)	980(++)	++(++)	++(++)	420(++)	++(++)	++(++)	++(++)	++(++)	++(++)	
Simazine	6.0(14)	4.7(6.4)	34(82)	190(460)	1.8(5.6)	20(66)	36(49)	30(58)	--	--	
Tebuthiuron	90(210)	70(96)	520(++)	++(++)	24(78)	370(++)	640(880)	550(++)	--	--	
Triclopyr	7.5(18)	5.9(8.0)	43(100)	240(580)	2.0(6.5)	24(76)	41(56)	35(67)	7.9(19)	30(65)	

Numbers in parentheses refer to margins of safety for workers wearing protective clothing.

MIX/L = Mixer/Loader

SUP = Supervisor

OBS = Observer

BP = Backpack

APPL = Sprayer

H&S = Hack and Squirt

INJ BAR = Injection Bar

++ = MOS of 1000 or more

-- = Not used in respective operation

Table 5-7

Worker Margins of Safety for Systemic Effects
Routine-Worst Case Scenarios

Herbicide	Aerial			Back Pack		Truck		Hand Application	
	P/LOT	MIX/L	SUP	OBS	BP	APPL	MIX/L	APPL MIX/L	H&S INJ BAR
Amitrole	3.7(8.9)	2.9(4.0)	22(51)	120(290)	-1.2(2.6)	5.9(19)	10(14)	8.7(17)	4.0(9.3) 15(33)
Asulam	110(210)	84(96)	620(++)	++(++)	29(77)	230(600)	390(450)	340(540)	-- --
Atrazine	5.5(13)	4.3(5.9)	32(76)	180(430)	1.5(4.8)	8.2(26)	14(20)	12(23)	-- --
Bromacil	--	--	--	--	1.0(3.2)	12(38)	20(28)	17(34)	5.0(12) 19(41)
2,4-D	2.5(5.9)	2.0(2.7)	14(34)	81(190)	-1.5(2.2)	7.7(25)	13(18)	11(22)	1.3(3.1) 4.9(11)
2,4-DP	19(45)	15(20)	110(260)	600(++)	2.9(9.4)	29(94)	51(70)	44(84)	4.1(9.7) 15(34)
Dalapon	9.0(21)	7.0(9.6)	52(120)	290(690)	2.0(6.5)	28(91)	49(67)	42(81)	-- --
Dicamba	75(178)	59(80)	430(++)	++(++)	20(64)	260(840)	450(620)	390(740)	40(94) 150(320)
Diuron	--	--	--	--	-5.9(-1.9)	-1.4(2.4)	1.3(1.8)	1.1(2.1)	-2.0(1.2) 1.9(4.1)
Fosamine	12(30)	9.8(13)	72(170)	400(960)	3.5(11)	44(140)	76(100)	65(130)	20(47) 74(160)
Glyphosate	37(89)	29(41)	220(510)	++(++)	10(32)	110(370)	208(280)	180(330)	-- --
Hexazinone	20(48)	16(21)	110(270)	650(++)	5.4(17)	31(100)	54(75)	47(90)	-- --
Picloram	170(420)	140(190)	++(++)	++(++)	59(190)	++(++)	++(++)	++(++)	230(550) 860(++)
Simazine	6.0(14)	4.7(6.4)	34(82)	190(460)	1.8(5.6)	20(66)	36(49)	30(58)	-- --
Tebuthiuron	12(30)	9.8(13)	72(170)	400(960)	3.4(11)	51(160)	89(120)	76(150)	-- --
Triclopyr	1.9(4.5)	1.5(2.0)	11(26)	60(140)	-2.0(1.6)	5.9(19)	10(14)	8.7(17)	2.0(4.7) 7.4(16)

Numbers in parentheses refer to margins of safety for workers wearing protective clothing.

MIX/L = Mixer/Loader

SUP = Supervisor

OBS = Observer

BP = Backpack

APPL = Sprayer

H&S = Hack and Squirt

INJ BAR = Injection Bar

++ = MOS of 1000 or more

-- = Not used in respective operation

Thus, workers would have to ignore their own safety and not wash the chemical off to receive doses as high as predicted in these accidents.

For workers who spill a pint of concentrate or spray mix on their skin there is a clear possibility that, with the exception of picloram, they could experience some acute toxic effects if they did not wash it off. The margins of safety for this accidental-worst case scenario are presented in Table 5-8. In the case of a spill of a pint of concentrate, many of the doses approach the LD₅₀. This represents a clear risk of severe toxic effects if the chemical is not washed off. There is some possibility that the damage caused by such a large acute dose could cause long-term damage to vital organs. There have also been rare instances in which limited exposure to 2,4-D was reported to cause permanent nerve damage. The dose and the risk are much greater for spills of concentrate than for the spray mix but, again, it is highly unlikely a worker would allow the chemical to penetrate his skin for any length of time. Attachment C presents the complete MOS and comparisons to LD₅₀'s for each herbicide.

Risk to Workers and the Public from Accidents Causing Large Spills of Herbicide

Table 5-9 summarizes the margins-of-safety for people drinking one liter of water contaminated by a large spill of herbicide from a helicopter or truck (see Attachment C, Tables C-145 through C-160). Most drinking water reservoirs would dilute the herbicide below no-observable-effect levels in a relatively short period of time. Both BLM and the Forest Service, in addition to EPA and the states, have procedures to minimize the risk to human health should a spill of this magnitude occur in or near a drinking water reservoir. Therefore, after the spill has been diluted, the risk to members of the public can be considered very low.

Spills into a small, stagnant pond would result in significantly higher doses and, in the event of a truck spill of 2,000 gallons would constitute a risk of chronic effects if members of the public continued to drink from it. Both the Forest Service and BLM have detailed spill prevention and clean up procedures to ensure that no member of the public is chronically exposed to a spill of this magnitude.

Probability of a Worst Case Accident

Some indication of the likelihood of significant herbicide spill accidents occurring may be derived from historical data. Herbicide spill accidents recorded by BLM and the Forest Service over 11 years were classified by location, date, and quantity spilled. Also included was information specifying the occurrence of accidents on the ground or in the air, and if the spill was near a waterway. Over an 11-year period, from 1973 through 1983, there were 24 recorded spills averaging 44.4 gallons per accident. Herbicide use rates ranged from 1.5 lb a.i. to 7 lb a.i. per acre for normal use rates. For a total of 302,085 acres sprayed during the 11-year period there was one accident for every 12,587 acres and 54 percent of the spills involved 30 gallons or less.

Table 5-8

Margins of Safety for
Spills onto the Skin of Workers Compared to Systemic
NOEL's and LD₅₀'s this Table updated

Herbicide	Spray Mix		Concentrate	
	NOEL	LD ₅₀	NOEL	LD ₅₀
Amitrole	-9.6	17,000	-48	3,400
Asulam	3.0	200	-4.0	17
Atrazine	-6.5	78	-65	7.8
Bromacil	-9.6	330	-38	17
2,4-D	-14	26	-140	2.6
2,4-DP	-1.9	55	-46	2.3
Dalapon	-4.0	130	--	--
Dicamba	2.1	63	-4.8	6.3
Diuron	-31	200	-380	16
Fosamine	-2.9	340	-9.6	100
Glyphosate	-1.0	140	-6.2	24
Hexazinone	-1.8	94	-12	14
Picloram	4.9	5,700	1.2	1,400
Simazine	-6.0	170	-48	21
Tebuthiuron	-2.9	18	--	--
Triclopyr	-19	13	-96	2.6

Table 5-9

Margins of Safety for People Drinking One Liter of Water
Contaminated by a Large Spill of Herbicide^a
Compared to the Systemic NOEL

Herbicide	Helicopter		Truck	
	Into a Reservoir	Into a Pond	Into a Reservoir	Into a Pond
Amitrole	11	-2.9	-1.8	-59
Asulam	31,000	980	1,600	49
Atrazine	1600	50	80	2.5
Bromacil	--	--	270	8.5
2,4-D	430	14	22	-1.5
2,4-DP	3,500	110	170	5.4
Dalapon	2,600	81	130	4.1
Dicamba	11,000	340	540	17
Diuron	--	--	17	-1.9
Fosamine	3,600	110	180	5.7
Glyphosate	10,000	340	1000	34
Hexazinone	5,800	180	290	9.1
Picloram	2,400	76	120	3.8
Simazine	1,700	54	87	2.7
Tebuthiuron	3,600	110	180	5.7
Triclopyr	540	17	27	-1.2

^aAssume a helicopter carrying 100 gallons of spray mix jettisons the entire load in a 16-acre by 8-foot-deep reservoir and a 1-acre by 4-foot-deep pond.

Table 5-10 shows the acreage sprayed, gallons spilled, and type of spill for the years 1973 to 1983. Figures 5-1 and 5-2 show that as the number of gallons increases, the probability of a spill decreases.

CANCER RISK

A worst-case analysis for cancer was conducted for the herbicides that had positive laboratory oncogenic studies (amitrole, asulam, atrazine, bromacil, and 2,4-DP) and for the herbicides (2,4-D, glyphosate, and picloram) for which there is scientific uncertainty. There is no evidence to suggest that any of the other herbicides could cause cancer. All of the other herbicides have negative cancer studies. EPA has requested additional data on the cancer potential of a number of the other 11 herbicides, and BLM and the Forest Service will consider the results of their findings when they become available.

Cancer is generally thought of as a nonthreshold response, which means a very small amount could cause a tumor and there is general agreement that amitrole has the potential to cause cancer in humans. In the case of amitrole, however, EPA has determined that the available evidence indicates a threshold carcinogenic response. A threshold response is consistent with the theory that amitrole is a secondary carcinogen because of its well-established anti-thyroid effects. Nevertheless, because there is some uncertainty about the mechanism of action of amitrole, a conservative approach has been taken in this analysis by assuming that amitrole's carcinogenicity is not a threshold effect. A threshold model would indicate zero or negligible carcinogenicity at low doses. This would be true of the log-probit model suggested by EPA (EPA, 1984s), but instead, the one-hit model has been used here under the assumption that even low doses may cause cancer. The one-hit model used for estimating the risk for all herbicides in this analysis predicts the maximum rates of cancer that could occur at low doses under any of the models that have been in general use. At high doses, all of the commonly used models would predict nearly the same rate of tumor formation.

Cancer risks for amitrole, asulam, atrazine, bromacil, 2,4-DP, 2,4-D, glyphosate, and picloram have been calculated based on a variety of conservative assumptions that are likely to overestimate the risks. These assumptions include the following:

1. Amitrole, asulam, bromacil, 2,4-DP, glyphosate, picloram, and 2,4-D are all assumed to be carcinogenic. Picloram and 2,4-D have not been shown conclusively to be carcinogenic in laboratory tests and there are many uncertainties about the glyphosate study, but the evidence also cannot show conclusively that they are not carcinogenic. Consequently, a worst case approach was taken.
2. In cases where there is more than one data set available, the data set indicating greater carcinogenic potency has been chosen. Carcinogenic potencies of 2,4-D and 2,4-DP have been calculated based on the rate of tumor formation in the female Osborne-Mendel rats studied by Hansen et al. (1971). This is the species and sex that have exhibited the highest rate of tumor formation after

Table 5-10

Number of Spills on Forest Service Land in
Washington and Oregon over the Last 10 Years

Number of Gallons	Number Spills (air and ground)	Avg. No. of Spills/ 1,000 Acres	Number Spills (air)	Avg. No. of Spills/ 1,000 Acres
0	24	0.0795	9	0.0298
10	19	0.0629	9	0.0298
20	14	0.0464	6	0.0199
30	11	0.0364	5	0.0166
40	11	0.0364	5	0.0166
50	10	0.0331	4	0.0132
60	8	0.0265	4	0.0132
70	6	0.0199	2	0.0066
80	6	0.0199	2	0.0066
90	3	0.0099	2	0.0066
100	1	0.0033	0	0.0000

2,4-D administration. All tumors were considered, although many of them were benign. Similarly for amitrole, the Food and Drug Research Laboratories rat study data (discussed in section 3) have been used.

3. It is assumed that carcinogenicity in all seven cases is not a threshold phenomenon: i.e., any dose of these chemicals has some probability of causing cancer, no matter how small the dose. This assumption is questionable for amitrole; EPA has determined that the evidence suggests a threshold phenomenon in this case.
4. The one-hit model was used to represent the relationship between dose and rate of tumor formation. This is the most conservative of several models that have been proposed because it predicts the highest cancer rates at the relatively low doses predicted for exposed humans. Other models that could have been used include the multistage, multihit, Weibull, logit, and probit models. The choice of model can affect the predicted cancer rates over several orders of magnitude. The one-hit model was used at one time by EPA, but the less conservative multistage model is now normally

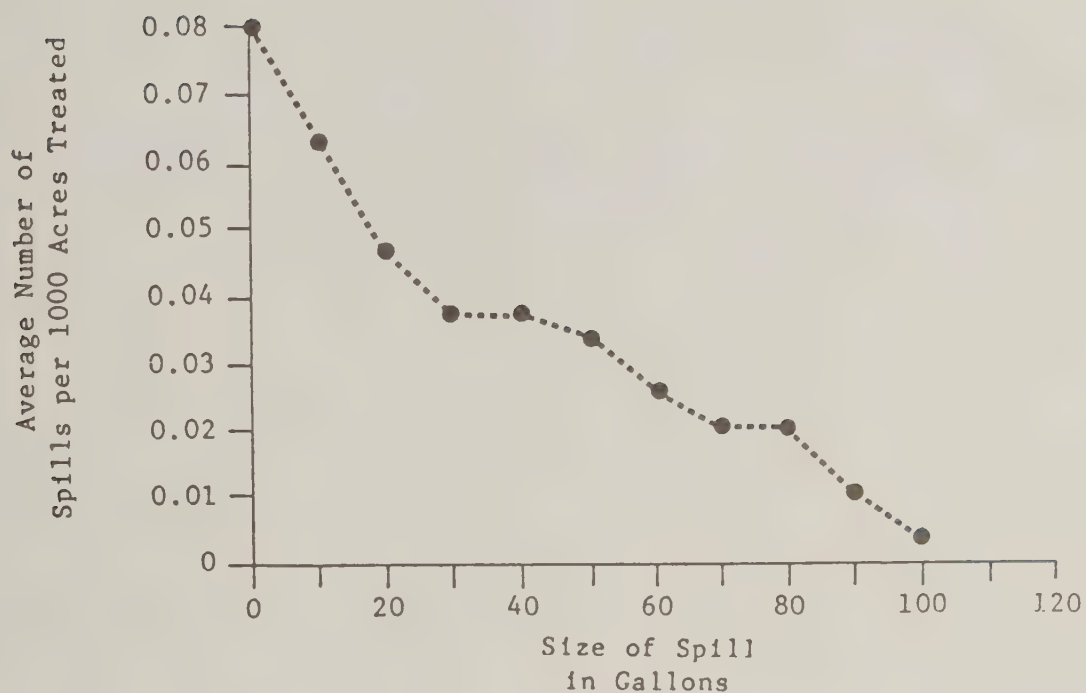


Figure 5-1 Frequency and Size of Spills from Air and Ground Operations

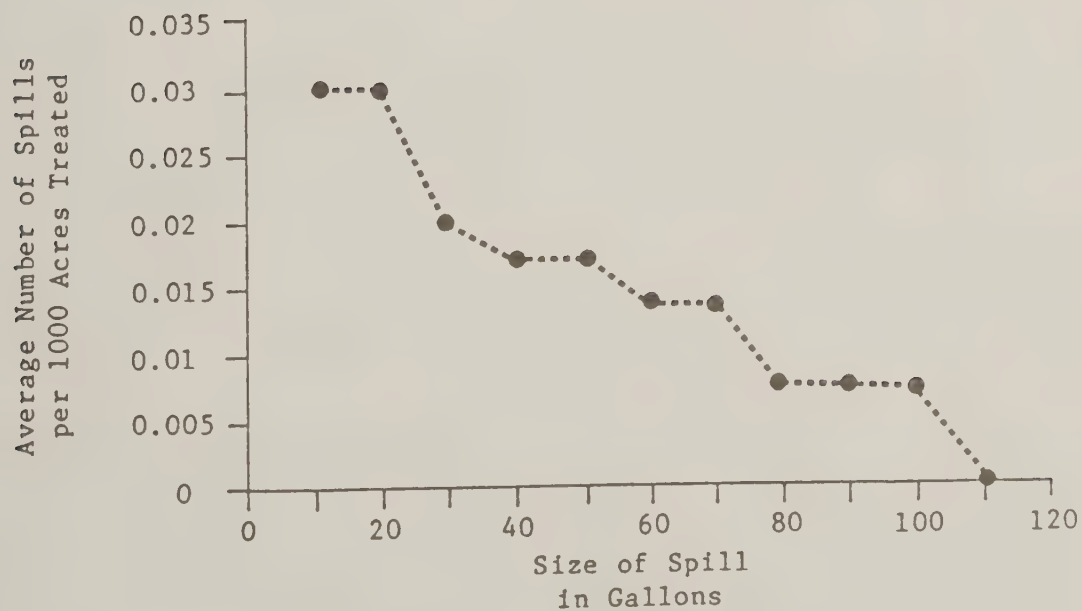


Figure 5-2 Frequency and Size of Spills from Air Operations

used. In the case of amitrole, EPA has suggested use of the even less conservative log-probit model.

5. In each case a 95-percent upper confidence limit was used to estimate cancer potency. For asulam, bromacil, 2,4-D and picloram these potencies were estimated using the maximum-likelihood procedure of the GLOBAL 82 computer program (Howe and Crump, 1982).
6. Interspecies extrapolation is a major source of uncertainty in judging cancer risk. The scaling method used in this analysis is the most conservative of the commonly accepted methods. The cancer potency of each chemical for humans was assumed to be the same as the potency for rats when scaled in terms of mg per m² of body surface area (mg/m²). This method is commonly used by EPA and others, but it is not the only acceptable approach. Another equally acceptable (OSTP, 1985) method is to scale doses in terms of mg per kg of body weight (mg/kg), resulting in estimates of cancer risk that are about 16 percent of those calculated here.
7. The range of doses calculated for workers and the public in the basic scenarios covers even extreme exposures that might be encountered with each application method. Unusual exposure situations, represented by accidental spraying and large herbicide spills, have also been considered.

The probability of occurrence of cancer over a lifetime as a result of exposure to each of the chemicals was calculated using the following equations:

$$P(d) = 1 - \exp(-K \times b \times d)$$
$$d = D \times N/L$$

where:

P(d) is a conservative estimate of the probability of cancer during a person's lifetime as the result of dose d.

d is the average daily dose over a lifetime (mg/kg/day)

K is an interspecies extrapolation factor

b is a conservative estimate for cancer potency in the test animal (derived in section 2).

The following cancer potencies (per mg/kg/day) were used: amitrole, 1.4; 2,4-DP, 5.9×10^{-2} ; asulam, 2×10^{-2} ; 2,4-D, 5.0×10^{-3} ; bromacil, 3.8×10^{-3} ; picloram, 5.7×10^{-4} ; and glyphosate, 2.4×10^{-5} .

D is the daily dose (mg/kg/day)

N is the number of days during which the dose D occurs during an individual's lifetime

L is the number of days in a lifetime, taken to be 25,550 for a 70-year life span.

The interspecies extrapolation factor, K, can be estimated by assuming that body surface area is proportional to body weight to the 2/3 power (Mantel and Schneiderman, 1975), so that K would be:

$$K = (\text{human weight/test animal weight})^{1/3}$$

For an average human weight of 50 kg and an average rat weight of 350 g, K is estimated to be 5.2.

Cancer Risk to the Public

Cancer risk for the general public has been calculated for a single exposure, and also for 30 exposures over a lifetime. The approximate cancer risks to the public for the routine-realistic and routine-worst case aerial scenarios are shown in Tables 5-11 and 5-12. (See Attachment C for the presentation of cancer risks to the public for the other four routine exposure scenarios). Of the seven chemicals, the greatest risks are shown for amitrole. In the highest exposure situation, the large aerial scenario, the maximum risk of cancer for a single exposure is less than 6 in 1 million, for a person eating vegetables from near the spray site. Even when several routes of exposure are added for the example nearby resident, the resulting risk for a single incident is still less than 10 in 1 million. The cumulative risk due to several exposures of this magnitude would be the sum of the risks for each exposure. The risk of cancer from 30 exposures is also given in the tables, but the chance of receiving 30 exposures as large as those in the worst case is negligible. If 30 routine-worst case exposures were experienced, the cumulative risk would be less than 3 in 10,000. Exposures in the routine-realistic cases lead to much lower risk. The risk of cancer due to doses from any of the routes of exposure in the typical aerial spraying scenario is less than 2 in 100,000, even for amitrole. Cancer risks for the other chemicals are very much less. The risk for glyphosate is never greater than 2 in 1 billion. For 2,4-D, 2,4-DP, asulam, and picloram, none of the routes of exposure in any scenario results in a cancer risk greater than about 2 in 10 million, per exposure. The highest risk of cancer to the public from bromacil usage is from backpack spraying of large areas because bromacil is not aerially applied in the forests of Region 6. The risk due to any routes of exposure is less than 4 in 100 million (Table C-162).

Cancer Risk to Workers

Cancer risk to workers has been calculated for an expected case assuming 5 years of employment in herbicide application, and an average number of days of spraying per year. The average number of exposures per lifetime was estimated to range from 30 to 70. The risk has been calculated in the extreme cases assuming 30 years of employment and a total of 288 to 480 exposures. It is very unlikely that a worker would apply herbicides on the

(insert Tables 5-11 and 5-12)

Table 5-11

Lifetime Cancer Risk^d--Exposed Public
Realistic Aerial, 40 Acres by Helicopter

Routes of Exposure	Exposures per Lifetime	Risk from Exclusive Use of:							
		2,4-D	2,4-DP	Asulam	Bromacil	Picloram	Amitrole	Glyphosate	Atrazine
<u>For a Single Exposure</u>									
Dermal, Spray	1	10 ^{-11^b}	10 ⁻¹⁰	10 ⁻¹²	— ^c	10 ⁻¹⁴	10 ⁻¹¹	10 ⁻¹³	10 ⁻¹⁰
Vegetation Contact									
Hiker	1	10 ⁻¹³	10 ⁻¹²	10 ⁻¹⁴	—	10 ⁻¹⁶	10 ⁻¹³	10 ⁻¹⁵	10 ⁻¹²
Picker	1	10 ⁻⁹	10 ⁻⁸	10 ⁻¹⁰	—	10 ⁻¹²	10 ⁻⁹	10 ⁻¹¹	10 ⁻⁸
Drinking Water	1	10 ⁻⁹	10 ⁻⁸	10 ⁻¹⁰	—	10 ⁻¹⁰	10 ⁻⁷	10 ⁻¹¹	10 ⁻⁸
Eating Berries	1	10 ⁻⁹	10 ⁻⁸	10 ⁻¹⁰	—	10 ⁻¹¹	10 ⁻⁷	10 ⁻¹¹	10 ⁻⁸
Eating Vegets.	1	10 ⁻⁹	10 ⁻⁸	10 ⁻¹⁰	—	10 ⁻¹⁰	10 ⁻⁷	10 ⁻¹¹	10 ⁻⁸
Eating Deer	1	10 ⁻¹⁰	10 ⁻⁹	10 ⁻¹¹	—	10 ⁻¹²	10 ⁻⁸	10 ⁻¹²	10 ⁻⁹
Eating Fish	1	10 ⁻¹⁰	10 ⁻⁹	10 ⁻¹⁰	—	10 ⁻¹¹	10 ⁻⁷	10 ⁻¹²	10 ⁻⁸
<u>Combined Routes of Exposure</u>									
Hiker	1	10 ⁻⁹	10 ⁻⁸	10 ⁻¹⁰	—	10 ⁻¹⁰	10 ⁻⁷	10 ⁻¹¹	10 ⁻⁸
Berry Picker	1	10 ⁻⁹	10 ⁻⁸	10 ⁻⁹	—	10 ⁻¹⁰	10 ⁻⁷	10 ⁻¹¹	10 ⁻⁸
Hunter	1	10 ⁻⁹	10 ⁻⁸	10 ⁻¹⁰	—	10 ⁻¹⁰	10 ⁻⁷	10 ⁻¹¹	10 ⁻⁸
Fisherman	1	10 ⁻⁹	10 ⁻⁸	10 ⁻¹⁰	—	10 ⁻¹⁰	10 ⁻⁷	10 ⁻¹¹	10 ⁻⁸
Resident	1	10 ⁻⁹	10 ⁻⁸	10 ⁻¹⁰	—	10 ⁻¹⁰	10 ⁻⁶	10 ⁻¹¹	10 ⁻⁸
<u>For 30 Exposures</u>									
Dermal, Spray	30	10 ⁻¹⁰	10 ⁻⁹	10 ⁻¹⁰	—	10 ⁻¹²	10 ⁻⁹	10 ⁻¹²	10 ⁻⁹
Vegetation Contact									
Hiker	30	10 ⁻¹²	10 ⁻¹¹	10 ⁻¹²	—	10 ⁻¹⁴	10 ⁻¹¹	10 ⁻¹⁴	10 ⁻¹¹
Picker	30	10 ⁻⁸	10 ⁻⁷	10 ⁻⁸	—	10 ⁻¹⁰	10 ⁻⁷	10 ⁻⁹	10 ⁻⁶
Drinking Water	30	10 ⁻⁸	10 ⁻⁷	10 ⁻⁸	—	10 ⁻⁹	10 ⁻⁵	10 ⁻¹⁰	10 ⁻⁷
Eating Berries	30	10 ⁻⁸	10 ⁻⁷	10 ⁻⁹	—	10 ⁻⁹	10 ⁻⁶	10 ⁻¹⁰	10 ⁻⁷
Eating Vegets.	30	10 ⁻⁸	10 ⁻⁷	10 ⁻⁸	—	10 ⁻⁹	10 ⁻⁵	10 ⁻¹⁰	10 ⁻⁷
Eating Deer	30	10 ⁻⁹	10 ⁻⁸	10 ⁻⁹	—	10 ⁻¹⁰	10 ⁻⁶	10 ⁻¹¹	10 ⁻⁸
Eating Fish	30	10 ⁻⁸	10 ⁻⁷	10 ⁻⁹	—	10 ⁻⁹	10 ⁻⁶	10 ⁻¹⁰	10 ⁻⁶
<u>Combined Routes of Exposure</u>									
Hiker	30	10 ⁻⁸	10 ⁻⁷	10 ⁻⁸	—	10 ⁻⁹	10 ⁻⁵	10 ⁻¹⁰	10 ⁻⁷
Berry Picker	30	10 ⁻⁷	10 ⁻⁶	10 ⁻⁸	—	10 ⁻⁹	10 ⁻⁵	10 ⁻⁹	10 ⁻⁶
Hunter	30	10 ⁻⁸	10 ⁻⁷	10 ⁻⁸	—	10 ⁻⁹	10 ⁻⁵	10 ⁻¹⁰	10 ⁻⁷
Fisherman	30	10 ⁻⁷	10 ⁻⁷	10 ⁻⁸	—	10 ⁻⁹	10 ⁻⁵	10 ⁻¹⁰	10 ⁻⁶
Resident	30	10 ⁻⁷	10 ⁻⁶	10 ⁻⁸	—	10 ⁻⁹	10 ⁻⁵	10 ⁻⁹	10 ⁻⁶

^aCancer risks shown in this Table were calculated based on a variety of assumptions that tend to overestimate risks as explained in Section 5.

^cNot used in aerial application.

^bAll of these numbers shown exponentially are to be interpreted as follows:

^dNot used in aerial application.

10⁻⁷ means 1 out of 10 million individuals exposed to a given herbicide via a given exposure scenario.

10⁻⁸ means 1 out of 100 million individuals,

10⁻⁹ means 1 out of 1 billion individuals, etc.

Table 5-12

Lifetime Cancer Risk^a--Exposed Public
Large Aerial, 400 Acres by Fixed Wing, Worst Case

Routes of Exposure	Exposures per Lifetime	Risk from Exclusive Use of:							
		2,4-D	2,4-DP	Asulam	Bromacil	Picloram	Amitrole	Glyphosate	$\frac{1}{2}$ Atrazine ³
<u>For a Single Exposure</u>									
Dermal, Spray	1	10 ^{-8^b}	10 ⁻⁸	10 ⁻⁹	— ^c	10 ⁻¹⁰	10 ⁻⁸	10 ⁻¹⁰	$\frac{1}{2}$ 10 ^{-7³/₄}
Vegetation Contact									
Hiker	1	10 ⁻¹⁰	10 ⁻⁹	10 ⁻¹¹	—	10 ⁻¹²	10 ⁻¹⁰	10 ⁻¹²	$\frac{1}{2}$ 10 ^{-9³/₄}
Picker	1	10 ⁻⁸	10 ⁻⁷	10 ⁻⁹	—	10 ⁻¹⁰	10 ⁻⁷	10 ⁻¹⁰	$\frac{1}{2}$ 10 ^{-7³/₄}
Drinking Water	1	10 ⁻⁸	10 ⁻⁸	10 ⁻⁹	—	10 ⁻⁹	10 ⁻⁶	10 ⁻¹⁰	$\frac{1}{2}$ 10 ^{-8³/₄}
Eating Berries	1	10 ⁻⁸	10 ⁻⁸	10 ⁻⁹	—	10 ⁻⁹	10 ⁻⁶	10 ⁻¹⁰	$\frac{1}{2}$ 10 ^{-8³/₄}
Eating Vegets.	1	10 ⁻⁸	10 ⁻⁷	10 ⁻⁹	—	10 ⁻⁹	10 ⁻⁶	10 ⁻¹⁰	$\frac{1}{2}$ 10 ^{-7³/₄}
Eating Deer	1	10 ⁻⁹	10 ⁻⁸	10 ⁻¹⁰	—	10 ⁻¹⁰	10 ⁻⁷	10 ⁻¹¹	$\frac{1}{2}$ 10 ^{-9³/₄}
Eating Fish	1	10 ⁻⁹	10 ⁻⁸	10 ⁻¹⁰	—	10 ⁻¹⁰	10 ⁻⁶	10 ⁻¹¹	$\frac{1}{2}$ 10 ^{-7³/₄}
<u>Combined Routes of Exposure</u>									
Hiker	1	10 ⁻⁸	10 ⁻⁷	10 ⁻⁹	—	10 ⁻⁹	10 ⁻⁶	10 ⁻¹⁰	$\frac{1}{2}$ 10 ^{-7³/₄}
Berry Picker	1	10 ⁻⁸	10 ⁻⁷	10 ⁻⁸	—	10 ⁻⁹	10 ⁻⁶	10 ⁻⁹	$\frac{1}{2}$ 10 ^{-7³/₄}
Hunter	1	10 ⁻⁸	10 ⁻⁷	10 ⁻⁹	—	10 ⁻⁹	10 ⁻⁶	10 ⁻¹⁰	$\frac{1}{2}$ 10 ^{-7³/₄}
Fisherman	1	10 ⁻⁸	10 ⁻⁷	10 ⁻⁹	—	10 ⁻⁹	10 ⁻⁶	10 ⁻¹⁰	$\frac{1}{2}$ 10 ^{-7³/₄}
Resident	1	10 ⁻⁸	10 ⁻⁷	10 ⁻⁹	—	10 ⁻⁹	10 ⁻⁶	10 ⁻¹⁰	$\frac{1}{2}$ 10 ^{-7³/₄}
<u>For 30 Exposures</u>									
Dermal, Spray	30	10 ⁻⁷	10 ⁻⁶	10 ⁻⁸	—	10 ⁻⁹	10 ⁻⁶	10 ⁻⁹	$\frac{1}{2}$ 10 ^{-6³/₄}
Vegetation Contact									
Hiker	30	10 ⁻⁹	10 ⁻⁸	10 ⁻⁹	—	10 ⁻¹¹	10 ⁻⁸	10 ⁻¹⁰	$\frac{1}{2}$ 10 ^{-8³/₄}
Picker	30	10 ⁻⁷	10 ⁻⁶	10 ⁻⁷	—	10 ⁻⁹	10 ⁻⁶	10 ⁻⁸	$\frac{1}{2}$ 10 ^{-6³/₄}
Drinking Water	30	10 ⁻⁷	10 ⁻⁶	10 ⁻⁸	—	10 ⁻⁸	10 ⁻⁴	10 ⁻⁹	$\frac{1}{2}$ 10 ^{-6³/₄}
Eating Berries	30	10 ⁻⁷	10 ⁻⁶	10 ⁻⁸	—	10 ⁻⁸	10 ⁻⁵	10 ⁻⁹	$\frac{1}{2}$ 10 ^{-6³/₄}
Eating Vegets.	30	10 ⁻⁷	10 ⁻⁶	10 ⁻⁷	—	10 ⁻⁸	10 ⁻⁴	10 ⁻⁹	$\frac{1}{2}$ 10 ^{-6³/₄}
Eating Deer	30	10 ⁻⁸	10 ⁻⁷	10 ⁻⁹	—	10 ⁻⁹	10 ⁻⁵	10 ⁻¹⁰	$\frac{1}{2}$ 10 ^{-7³/₄}
Eating Fish	30	10 ⁻⁷	10 ⁻⁶	10 ⁻⁸	—	10 ⁻⁸	10 ⁻⁵	10 ⁻⁹	$\frac{1}{2}$ 10 ^{-6³/₄}
<u>Combined Routes of Exposure</u>									
Hiker	30	10 ⁻⁷	10 ⁻⁶	10 ⁻⁷	—	10 ⁻⁸	10 ⁻⁴	10 ⁻⁸	$\frac{1}{2}$ 10 ^{-6³/₄}
Berry Picker	30	10 ⁻⁶	10 ⁻⁵	10 ⁻⁷	—	10 ⁻⁷	10 ⁻⁴	10 ⁻⁸	$\frac{1}{2}$ 10 ^{-5³/₄}
Hunter	30	10 ⁻⁷	10 ⁻⁶	10 ⁻⁷	—	10 ⁻⁸	10 ⁻⁴	10 ⁻⁸	$\frac{1}{2}$ 10 ^{-6³/₄}
Fisherman	30	10 ⁻⁷	10 ⁻⁶	10 ⁻⁷	—	10 ⁻⁸	10 ⁻⁴	10 ⁻⁸	$\frac{1}{2}$ 10 ^{-5³/₄}
Resident	30	10 ⁻⁶	10 ⁻⁵	10 ⁻⁷	—	10 ⁻⁷	10 ⁻⁴	10 ⁻⁸	$\frac{1}{2}$ 10 ^{-6³/₄}

^aCancer risks shown in this Table were calculated based on a variety of assumptions that tend to overestimate risks as explained in Section 5.

^bAll of these numbers shown exponentially are to be interpreted as follows:

10⁻⁷ means 1 out of 10 million individuals exposed to a given herbicide via a given exposure scenario.

10⁻⁸ means 1 out of 100 million individuals,

10⁻⁹ means 1 out of 1 billion individuals, etc.

^cNot used in aerial application.

number of days assumed in the worst case. The lifetime cancer risks for workers are shown in Table 5-13. (Cancer risk to workers for the accidental-worst case scenario is shown in Attachment C.) The risks for each herbicide were calculated assuming that only that herbicide was used. The highest risks for workers involve 2,4-DP use. The lifetime cancer risk to a backpack sprayer using only 2,4-DP is about 2 in 10,000 in the expected case. In the worst case the risk is greater than 1 in 1,000. The risk is much less for the other chemicals. The highest risk for 2,4-D is about 1 in 100,000 for backpack spraying in the expected case, and in the extreme case the greatest risk is about 1 in 10,000. The risk is somewhat greater for amitrole: as high as 7 in 100,000 for the realistic backpack exposure. Workers using asulam and bromacil in the extreme case have a lifetime cancer risk of less than 6 in 100,000 in all worker categories. The cancer risk from picloram or glyphosate use is even less for all worker categories. The risk in the expected case never exceeds 3 in 10 million.

EPA (1985a) has also conducted a carcinogenic risk assessment for workers using amitrole (see Table 5-14). EPA assumed that workers wore no protective clothing, and the estimated exposures were only 1.5×10^{-3} mg/kg/day for the highest exposures. EPA estimated cancer potency for both liver and thyroid tumors, using the log-probit and multistage models. Estimated cancer risks for the anti-thyroid action of amitrole were all less than 1 in 10 billion based on the log-probit model. The multistage model gave much higher risk estimates, especially for liver tumors. Calculated risks for average worker exposures were not greater than 1 in 10,000, and for maximum exposures the risks were not greater than 1 in 1,000. Amitrole carcinogenicity risks calculated in this risk assessment are consistent with EPA's independently derived results.

Cancer Risks in Accidental Situations

Cancer risks calculated for exposures due to accidental spraying are shown in Attachment C (Table C-165). The greatest risks among the seven chemicals are for amitrole. A single incident of accidental spraying of amitrole gives calculated risks of 6 in 100,000 for a person eating sprayed vegetables and 3 in 100,000 for a person drinking sprayed water. Among the other chemicals, the greatest risks are about 4 in 1 million for exposures to 2,4-DP, and 3 in 10 million for 2,4-D. Multiple incidents could be expected to result in cumulative risks. Cancer risks calculated for spill situations are also shown in Attachment C. The greatest risks are for spills of herbicide concentrate directly onto clothing and skin. Workers are at the greatest risk for this type of accident. The tabled values assume that most of a person's skin has been contacted by the solution, and cleanup does not occur for several hours. This is certainly contrary to standard practice. A spill of 2,4-DP concentrate onto a person gives a risk of about 3 in 1,000, and a spill of spray mixture gives a lesser risk of about 1 in 10,000. The corresponding risks for 2,4-D and amitrole are about a factor of 10 less. The risk of cancer due to spills of asulam and bromacil is about 1 in 100,000 for the concentrate and 1 in 1 million for the spray mixture. A spill of picloram or glyphosate concentrate gives a risk of 2 in 1 million or less. Cancer risks arising from even major spills into drinking water supplies are significantly less. A 100-gallon helicopter load of amitrole spray mixture dumped into a 1-acre pond would

(insert Table 5-13)

Table 5-13
Lifetime Cancer Risk^a--Exposed Workers

Routes of Exposure	Exposures per Lifetime	Risk from Exclusive Use of:							
		2,4-D	2,4-DP	Asulam	Bromacil	Picloram	Amitrole	Glyphosate	Atrazine
For Realistic Number of Exposures ^b									
Pilot	30	10 ⁻⁶	10 ⁻⁵	10 ⁻⁷	--d	10 ⁻⁸	10 ⁻⁶	10 ⁻⁸	10 ⁻⁵
Mixer-Loader	30	10 ⁻⁶	10 ⁻⁵	10 ⁻⁷	--	10 ⁻⁸	10 ⁻⁶	10 ⁻⁸	10 ⁻⁵
Supervisor	30	10 ⁻⁷	10 ⁻⁶	10 ⁻⁸	--	10 ⁻⁹	10 ⁻⁷	10 ⁻⁹	10 ⁻⁶
Observer	30	10 ⁻⁸	10 ⁻⁷	10 ⁻⁸	--	10 ⁻¹⁰	10 ⁻⁷	10 ⁻¹⁰	10 ⁻⁷
Backpack	50	10 ⁻⁵	10 ⁻⁴	10 ⁻⁶	10 ⁻⁶	10 ⁻⁸	10 ⁻⁵	10 ⁻⁷	10 ⁻⁴
R-O-W Sprayer	45	10 ⁻⁷	10 ⁻⁶	10 ⁻⁷	10 ⁻⁷	10 ⁻⁹	10 ⁻⁶	10 ⁻⁸	10 ⁻⁶
R-O-W Mix/L	45	10 ⁻⁷	10 ⁻⁶	10 ⁻⁷	10 ⁻⁷	10 ⁻⁹	10 ⁻⁶	10 ⁻⁹	10 ⁻⁶
R-O-W AP/M/L	45	10 ⁻⁷	10 ⁻⁶	10 ⁻⁷	10 ⁻⁷	10 ⁻⁹	10 ⁻⁶	10 ⁻⁹	10 ⁻⁶
Hack and Squirt	70	10 ⁻⁶	10 ⁻⁴	--3	10 ⁻⁶	10 ⁻⁸	10 ⁻⁵	--	--
Injection Bar	70	10 ⁻⁶	10 ⁻⁵	--	10 ⁻⁷	10 ⁻⁸	10 ⁻⁶	--	--
Worst Case Number of Exposures									
Pilot	288	10 ⁻⁵	10 ⁻⁴	10 ⁻⁶	--	10 ⁻⁸	10 ⁻⁵	10 ⁻⁷	10 ⁻⁴
Mixer-Loader	288	10 ⁻⁵	10 ⁻⁴	10 ⁻⁶	--	10 ⁻⁷	10 ⁻⁵	10 ⁻⁷	10 ⁻⁴
Supervisor	288	10 ⁻⁶	10 ⁻⁵	10 ⁻⁷	--	10 ⁻⁸	10 ⁻⁶	10 ⁻⁸	10 ⁻⁵
Observer	288	10 ⁻⁷	10 ⁻⁶	10 ⁻⁷	--	10 ⁻⁹	10 ⁻⁶	10 ⁻⁹	10 ⁻⁶
Backpack	440	10 ⁻⁴	10 ⁻³	10 ⁻⁵	10 ⁻⁵	10 ⁻⁷	10 ⁻⁴	10 ⁻⁶	10 ⁻³
R-O-W Sprayer	416	10 ⁻⁶	10 ⁻⁵	10 ⁻⁶	10 ⁻⁶	10 ⁻⁸	10 ⁻⁵	10 ⁻⁸	10 ⁻⁵
R-O-W Mix/L	416	10 ⁻⁶	10 ⁻⁵	10 ⁻⁶	10 ⁻⁶	10 ⁻⁸	10 ⁻⁵	10 ⁻⁸	10 ⁻⁵
R-O-W AP/M/L	416	10 ⁻⁶	10 ⁻⁵	10 ⁻⁶	10 ⁻⁶	10 ⁻⁸	10 ⁻⁵	10 ⁻⁸	10 ⁻⁵
Hack and Squirt	480	10 ⁻⁵	10 ⁻⁴	--	10 ⁻⁵	10 ⁻⁷	10 ⁻⁴	--	--
Injection Bar	480	10 ⁻⁵	10 ⁻⁴	--	10 ⁻⁶	10 ⁻⁸	10 ⁻⁵	--	--

^aCancer risks shown in this Table were calculated based on a variety of assumptions that tend to overestimate risks as explained in Section 5.

^bAll of these numbers shown exponentially are to be interpreted as follows:

10⁻⁷ means 1 out of 10 million individuals exposed to a given herbicide via a given exposure scenario.

10⁻⁸ means 1 out of 100 million individuals,

10⁻⁹ means 1 out of 1 billion individuals, etc.

^cNot used in hand application.

^dNot used in aerial application.

Table 5-14

Amitrole Worker Exposure Estimates
and Related Estimates of Cancer Risk

Exposure Situation ^a	Exposure (mg/kg/day)	Log- Probit ^b	Upper 95% Bound on Risk	
			Multi-Stage	
			Q _{1e} * = .20 ^c	Q _{1u} * = .076 ^d
<u>Utility Power Wagon Mixer-Loader/Applicator</u>				
Minimum	1.1x10 ⁻⁴ ^e	10-10	10 ⁻⁵	10 ⁻⁵
Average	4.6x10 ⁻⁴	10-10	10 ⁻⁴	10 ⁻⁵ - 10 ⁻⁴
Maximum	1.2x10 ⁻³	10-10	10 ⁻⁴	10 ⁻⁴
<u>Industry Power Wagon Mixer-Loader/Applicator</u>				
Minimum	6.3x10 ⁻⁵	10-10	10 ⁻⁵¹	10 ⁻⁶ - 10 ⁻⁵
Average	4.3x10 ⁻⁴	10-10	10 ⁻⁴	10 ⁻⁵ - 10 ⁻⁴
Maximum	1.1x10 ⁻³	10-10	10 ⁻⁴	10 ⁻⁴
<u>Industry Knapsack/ Hand Carry Applicator</u>				
Minimum	4.9x10 ⁻⁶	10-10	10 ⁻⁶	10 ⁻⁷ - 10 ⁻⁶
Average	3.7x10 ⁻⁴	10-10	10 ⁻⁵ - 10 ⁻⁴	10 ⁻⁵
Maximum	1.5x10 ⁻³	10-10	10 ⁻⁴ - 10 ⁻³	10 ⁻⁴
<u>Industry Knapsack/ Hand Carry/Mixer-Loader</u>				
Minimum	5.3x10 ⁻⁷	10-10	10 ⁻⁷	10 ⁻⁸ - 10 ⁻⁷
Average	2.7x10 ⁻⁵	10-10	10 ⁻⁶ - 10 ⁻⁵	10 ⁻⁶
Maximum	1.3x10 ⁻⁴	10-10	10 ⁻⁵	10 ⁻⁵

^aExposure estimates of dermal exposure and inhalation exposure were taken from Hitch memo of 12/15/83 adjusted for a maximum of 0.1 percent dermal penetration. Zendzian memo of 6/26/85, annual exposure estimate is divided by 70 kg to obtain exposure in mg/kg and by 365 and 2 to obtain average daily dose for one-half of a 70-year lifetime.

^bLog-Probit: This column represents the risks bounds under the assumption of the anti-thyroid action of amitrole.

^c Q_{1e}^* : This column represents the risk bounds under the assumption of the interspecies surface area correction; and based on liver tumors in female mice.

^d Q_{1u}^* : This column represents the risk bounds without the interspecies surface area correction; and based on thyroid tumors in male rats.

^eCancer risk shown in this Table were calculated based on a variety of assumptions that tend to overestimate risks as explained in Section 5.

lead to a risk of cancer of not more than 2 in 100,000 for a person drinking a liter of the water. The corresponding risks for the other chemicals are very much less. If a 1,000-gallon tank truck of spray mixture were spilled into a small pond, the risk for amitrole would be about 2 in 10,000. For 2,4-D the corresponding cancer risk is less than 1 in 1 million.

Comparison of Cancer Risks with Other Common Risks

To put the cancer risks calculated here in perspective, Table 5-15 lists risks due to some more familiar hazards and occupational risks. Motor vehicle accidents have a risk of fatality that averages 2 in 10,000 per person each year. Over a 30-year period, the cumulative risk would be 6 in 1,000. A variety of hazards are listed in the table that have a risk of about 1 in 1 million. They include smoking 2 cigarettes, eating 6 pounds of peanut butter, drinking 40 sodas sweetened with saccharin, or taking one transcontinental round trip by air. The cancer risk for a single x ray is 7 in 1 million. Many occupational risks are greater. Working for 30 years in agriculture or construction has a risk of about 1.8 in 100, and in mining and quarrying the risk is even greater: 3 in 100 over 30 years.

RISK OF HERITABLE MUTATIONS

No human studies are available that associate any of the herbicides with heritable mutations. Furthermore, no risk assessments that quantify the probability of mutations are available in the literature or from EPA. Laboratory studies constitute the best available information on mutagenic potential. Results of the mutagenicity assays conducted on the 16 herbicides are summarized in Table 3-3.

For some of the herbicides, no validated mutagenicity tests exist or the mutagenicity tests conducted are insufficient to conclude whether the chemical is mutagenic. For these herbicides, a worst case assumption is made that these herbicides have the potential to cause mutations in humans. In these cases the results of carcinogenicity tests (see Table 3-3) or cancer risk assessments can be used to estimate the risk of heritable mutations. The rationale for this assumption is summarized by the USDA (1985) as follows: "Since mutagenicity and carcinogenicity both follow similar mechanistic steps (at least those that involve genetic toxicity), the increased risk of cancer can be used to approximate the quantitative risk of heritable mutations. The basis for this assumption is that both mutagenicity and at least primary carcinogens react with DNA to form a mutation or DNA lesion affecting a particular gene or set of genes. The genetic lesions then require specific metabolic processes to occur, or the cells must divide to insert the lesion into the genetic code of the cell. We believe the cancer risk provides a worst case approximation to heritable mutations because cancer involves many types of cells whereas heritable mutations involve only germinal (reproductive) cells."

(insert Table 5-15)

Table 5-15

Lifetime Risk of Death or Cancer Resulting from Everyday Activities

Activity	Need to Accumulate a One in a Million Risk of Death	Average Annual Risk ^a per Capita
Based on living in the United States		
Motor vehicle accident	1.5 days	2×10^{-4} ^b
Falls	6 days	6×10^{-5}
Drowning	10 days	4×10^{-5}
Fires	13 days	3×10^{-5}
Firearms	36 days	1×10^{-5}
Electrocution	2 months	5×10^{-6}
Tornados	20 months	6×10^{-7}
Floods	20 months	6×10^{-7}
Lightning	2 years	5×10^{-7}
Animal bite or sting	4 years	2×10^{-7}
Occupational Risks		
General		
manufacturing	4.5 days	8×10^{-5}
trade	7 days	5×10^{-5}
service and government	3.5 days	1×10^{-4}
transport and public utilities	1 day	4×10^{-4}
agriculture	15 hours	6×10^{-4}
construction	14 hours	6×10^{-4}
mining and quarrying	9 hours	1×10^{-3}
Specific		
coal mining (accidents)	14 hours	6×10^{-4}
police duty	1.5 days	2×10^{-4}
railroad employment	1.5 days	2×10^{-4}
fire fighting	11 days	8×10^{-4}

Table 5-15 (Cont.)

Activity	Need to Accumulate a One in a Million Risk of Death	Average Annual Risk ^a per Capita
Everyday Risks		
Eating and drinking	40 diet sodas (saccharin) 6 pounds of peanut butter (aflatoxin) 180 pints of milk (aflatoxin) 200 gallons of drinking water from Miami or New Orleans 90 pounds of broiled steak (cancer risk only)	
Smoking	2 cigarettes	

^aNote to calculate the risk over a lifetime multiply this column by 70. From Crouch and Wilson (1982).

^bAll of these numbers shown exponentially are to be interpreted as follows:
 10^{-7} means 1 out of 10 million individuals exposed to a given herbicide via a given exposure scenario.
 10^{-8} means 1 out of 100 million individuals,
 10^{-9} means 1 out of 1 billion individuals.

Asulam and glyphosate tested negative for mutagenicity in all assays conducted, and thus can be considered to pose no mutagenic risk. Fosamine, hexazinone, simazine, and triclopyr were nonmutagenic in the great majority of assays conducted and were nononcogenic in all of the carcinogenicity tests performed; therefore, it can be assumed that their mutagenic risk is slight to negligible. Dicamba was nonmutagenic in most of the assays performed and no oncogenicity was found in several long-term studies. EPA (1985d) has classified the chronic studies as "inadequate to evaluate the oncogenic potential of dicamba." Due to the bulk of negative results, dicamba can be considered as a mutagen in the worst case analysis but the mutagenic hazard would be extremely limited.

No validated mutagenicity studies have been conducted with dalapon, or diuron. The worst case assumption is that all of these chemicals are mutagenic. The probability of dalapon or diuron causing heritable mutations is low because they have not been shown to cause cancer in any long-term studies.

The negative oncogenic studies for diuron were classified by EPA (1985d) as inadequate to determine carcinogenic potential to mammalian organisms. The lack of positive results in mutagenic or oncogenic tests with diuron suggests that diuron would present a very low risk to humans as a mutagen.

Bromacil tested positive in one of two oncogenic studies. The risk of heritable mutations from the chemical should be no greater than the estimates of cancer risk based on a worst case approximation.

Atrazine tested positive for mutagenicity in 15 of 33 assays. The worst case assumption is that atrazine is mutagenic. However, many of the positive results were achieved through tests that may not be relevant to evaluating mutagenic risk in humans. Some positive results in rodents were also achieved, but these in vivo responses were only observed at levels greater than 1,500 mg/kg body weight. These are exceptionally high levels and suggest that the degree of germ cell hazard from low levels of atrazine would be minimal. The worst case estimate for atrazine mutagenic effects would be the risk of cancer as shown in Tables 5-11 through 5-13.

Amitrole was nonmutagenic in 56 microbial gene mutation tests. The results of two tests which were positive are considered of questionable validity by EPA (1985a), and overall it is considered to pose no potential for heritable mutations (EPA, 1985a). The worst-case estimate for amitrole mutagenic effects would be the risk of cancer as shown in Tables 5-11 and 5-12.

For picloram and 2,4-D, there have been only a few studies performed and these have indicated both positive and negative mutagenic potential. EPA has requested more mutagenicity-test information for both of these compounds. A number of comprehensive reviews of the 2,4-D mutagenic data have indicated that it does not pose significant risk of human gene mutations (USDA, 1984). 2,4-D has been shown to be nononcogenic in the two carcinogenicity studies that have been conducted. Based on a worst case estimate, the risk of heritable mutations from these chemicals would be no greater than the estimates of cancer risk.

Mutagenic tests with 2,4-DP have shown mixed results. 2,4-DP was negative in four microbial assays and positive in four other assays; therefore it

may have limited genotoxic potential. Based on the limited test data presented in Section 3, one cannot presume mutagenic hazard because no in vivo or mammalian assays have been conducted. However, the worst case assumption is that 2,4-DP is mutagenic and the mutagenic risk in the worst case would be no greater than the risk of cancer.

SYNERGISTIC AND CUMULATIVE EFFECTS

Synergistic Effects

Synergistic effects of herbicides are those that occur because of simultaneous exposure to more than one herbicide and that cannot be predicted based on the effects of the individual chemicals. A synergistic effect occurs when the combined effect of two chemicals is much greater than the sum of the effects of each agent given alone. Based on the limited amount of data available on pesticide combinations, it is possible but very unlikely that synergistic effects could occur as a result of exposure to two or more of the herbicides considered in this analysis.

The effects of many of the possible herbicide combinations have not been studied. This is not surprising because the first priority must be to study the effects of the herbicides individually, and this type of information is not yet sufficient in some cases. Moreover, the combinations that could be studied are too numerous to be listed. The combinations of interest include not only combinations of 2 or more of the 16 herbicides, but also combinations of the herbicides with other chemicals, such as insecticides, that exist in the environment.

However, Kociba and Mullison (1985) in describing toxicological interactions with agricultural chemicals state:

Our present scientific knowledge in toxicology indicates that an exposure to a mixture of pesticides is more likely to lead to additivity or antagonism rather than synergism when considering the toxicological effects of such a combination. To be conservative and for reasons of safety, an additive type of toxicological response is generally assumed rather than an antagonistic type of response.

In the case of registered pesticides, a great amount of toxicological information is developed during the research and development of each individual pesticide. In addition to this information on individual pesticides, short term toxicity studies are always done prior to the selling of a pesticide mixture. Should synergism unexpectedly be present in a proposed commercial mixture of two pesticides, it would be identified in such cases and would then be dealt with accordingly. In toxicological tests involving a combination of commercial pesticides, synergism has generally not been observed.

Kociba and Mullison (1985) use a specific example of a mixture of 2,4-D and picloram to illustrate their point. They list the LD₅₀'s of each herbicide separately and the LD₅₀ of the mixture. The mixture LD₅₀ is between the LD₅₀'s of the two constituents indicating lack of a synergistic effect.

The possibility of synergistic effects can be examined for the 16 herbicides addressed in this analysis in the same way (see Table 5-16). Based on a review of oral acute LD₅₀'s for mixtures of 2 or more of the 16 herbicides used in this analysis that have been submitted to EPA, no synergistic effects were found. In no case is the mixture of two herbicides more acutely toxic than any of the constituents. For example, a mixture of 2,4-D and Banvel 45 (dicamba) formulation resulted in a NOEL of 1,847 mg/kg, with lower acute LD₅₀ values reported for the technical grade of both formulation constituents (EPA, 1984e). Rat inhalation studies of formulations containing 2,4-D and picloram did not result in toxic effects (EPA, 1984q). This gives at least a first indication that synergistic effects are unlikely to occur in the vegetation management program.

One synergistic effect documented in the literature is the combination of 2,4-D and picloram produces skin sensitization, while neither alone produces this effect. (EPA, 1984q). Moreover, it is highly unlikely that synergistic adverse effects could result from exposure to more than one herbicide applied in separate projects. There are several reasons for this. First, unlike the situation in conventional agriculture, herbicide residues in plants and soil are not expected to persist from one application to another, even for the more persistent herbicides. Silvicultural and range applications are not annual; there are typically many years between applications.

Second, none of the 16 herbicides accumulates in human tissues, so exposure of an individual to 2 herbicides at different times would be unlikely to cause simultaneous residues within the body.

Third, exposures to the herbicides, especially for the public, are normally quite small. The greater exposures considered in the routine-worst case scenarios would occur only very infrequently, and the probability of the accidental exposures is extremely low. Because the probability of a large exposure is small for any one chemical, the probability of large exposures simultaneously to multiple chemicals is negligible. This is because the probability of two independent events occurring simultaneously is the product of the probabilities of the individual events. For example, if the probability of a person receiving a given exposure is 1 in 1,000 for 2 herbicides, then the probability of receiving that exposure to both herbicides would be 1 in 1 million.

Simultaneous exposure to more than one chemical is likely in cases where those chemicals are combined in a single spray mixture. Although the great majority of vegetation control projects in the Region involve only a single herbicide, a number of acres are treated with mixtures of herbicides. However, the only herbicide mixtures used are combinations that have been approved for use by the Environmental Protection Agency.

Cumulative Effects

The total area of U.S. Forest Service land in Washington and Oregon and BLM land in western Oregon is 38,000 square miles. This area makes up about one-fourth of the total land area (165,000 mi²) of those two States. In

(insert Table 5-16)

Table 5-16.
Acute LD₅₀'s of Herbicide Mixtures Compared to Acute LD₅₀'s of Mixtures

Combination	Test	Level in Comp. 1	Level in Comp. 2	Level in Comp. 3	Level in Comp. 4
Banvel 4	Acute Dermal rabbit	Banvel 4 (Dicamba) rabbit (level not given)	Atrazine 80 WP (level not given)	Princip WP Simazine (level not given)	Paraquat 2EC (level not given)
Atrazine 80 WP	LD ₅₀ 20,000 mg/kg	LD ₅₀ 2,000 mg/kg Tech., DMA Salt	LD ₅₀ 7,000 kg/mg Tech., rabbit	BOW formulation rabbit	LD ₅₀ 24 mg/kg (W.S.S.A)
Princip. WP					
Paraquat 2EC					
Banvel 4	Acute Oral rat	Banvel 4	Atrazine 80 WP	Princip. WP (Simazine)	Paraquat 2EC
Atrazine 80 WP	(level not given)				
Princip. WP	LD ₅₀ 5,000 mg/kg	LD ₅₀ 757 mg/kg	LD ₅₀ 1,869 kg/mg	LD ₅₀ 5,000 kg/mg	LD ₅₀ 120 mg/kg (W.S.S.A)
Paraquat 2EC					
2,4-D	Acute Oral rat	2,4-D 71.42% 532 mg/kg	MCPA .71% 800 kg/mg	Dicamba .04% 757 kg/mg rat	
MCPA	LD ₅₀ 5,000 mg/kg				
Dicamba					
2,4-D	Acute Dermal rabbit	2,4-D	MCPA	Dicamba	
MCPA	LD ₅₀ 2,000 mg/kg	1,400 mg/kg (DOE 1983)		LD ₅₀ 2,000 mg/kg	
Dicamba					
2,4-D	Acute Oral rat	2,4-D	Banvel 45		
and	LD ₅₀ 1,847 mg/kg	LD ₅₀ 532 mg/kg	LD ₅₀ 757 mg/kg		
Banvel 45					
(Dicamba)					
Levels not given					
2,4-D	Acute Dermal rabbit	2,4-D	Banvel 45		
and	LD ₅₀ 11,892 mg/kg	LD ₅₀ 400 mg/kg (DOE 1983)	LD ₅₀ 2,000 mg/kg		
Banvel 45					
(Dicamba)					
Levels not given					
2,4-D	Acute Oral	2,4-D 1.15% 532 mg/kg	Dicamba 1.60% 800 kg/mg	MCP 1.06% (mecoprop)	
1.15%				W.S.S.A.	
Dicamba	10 LD ₅₀ 20 gm slight erythrae edema				
1.60%					
MCP					
2,4-D	Acute Dermal	2,4-D .99% 1,400 mg/kg (DOE 1983)	MCP 900 mg/kg (W.S.S.A.)		
.99%	LD ₅₀ 2,000 mg/kg				
MCP					

Table 5-16 (continued).

Combination	Test	Level in Comp. 1	Level in Comp. 2	Level in Comp. 3	Level in Comp. 4
2,4-D	Acute Oral	2,4-D			
MCPP	LD50 5,050 mg/kg	.582%	MCPP	.2448%	Dicamba .0516%
Dicamba			LD50 532 mg/kg	LD50 1,060 mg/kg	LD50 757 mg/kg
			(W.S.S.A.)	(W.S.S.A.)	
Banvel 45	Acute Oral	Banvel 45	Lasso 4EC		
+ Lasso 4EC	LD50 5,000 mg/kg	LD50 757 mg/kg	(Alachlor)		
(Alachlor)			1,000 mg/kg		
% comp. not given			(W.S.S.A.)		
Banvel 45	Acute Dermal	Banvel 45	Lasso 4EC		
+ Lasso 4EC	LD50 20,000 mg/kg	(Dicamba)	(Alachlor)		
(Alachlor)		LD50 2,000 mg/kg	13,300 mg/kg		
% comp. not given			(W.S.S.A.)		
2,4-D	Acute Dermal,	2,4-D	MCPP	.612%	Dicamba .129%
MCPP	rabbit	LD50 1,400 mg/kg	LD50 2,000 mg/kg	LD50 2,000 mg/kg	
Dicamba	LD50 2,005 mg/kg	(DOE 1983)			
2,4-D	Acute Oral	2,4-D	MCPP	.680%	Dicamba .027%
MCPP	LD50 5,000 mg/kg	LD50 532 mg/kg	LD50 1,060 mg/kg	LD50 757 mg/kg	
Dicamba			(WSSA 1983)		
2,4-D	Acute Dermal	2,4-D	MCPP	.680%	Dicamba .027%
MCPP	LD50 2,000 mg/kg	LD50 1,400 mg/kg	LD50 900 mg/kg	LD50 2,000 mg/kg	
Dicamba		(DOE 1983)	(WSSA 1983)		
2,4-D	Acute Oral	2,4-D	MCPP	1.37%	Dicamba .055%
MCPP	LD50 5,000 mg/kg	LD50 532 mg/kg	LD50 1,060 mg/kg	LD50 2,000 mg/kg	
Dicamba			(WSSA 1983)		
2,4-D	Acute Dermal,	2,4-D	MCPP	1.37%	Dicamba .055%
MCPP	rabbit	LD50 1,400 mg/kg	LD50 900 mg/kg	LD50 2,000 mg/kg	
Dicamba	LD50 2,000 mg/kg	(DOE 1983)	(WSSA 1983)		
2,4-D	Acute Oral,	2,4-D	MCPP	.58%	
MCPP	rat	LD50 532 mg/kg	LD50 1,060 mg/kg		
	LD50 5,000 mg/kg		(WSSA 1983)		
2,4-D	Acute Dermal,	2,4-D	MCPP	.58%	
MCPP	rabbit	LD50 1,400 mg/kg	LD50 900 mg/kg		
Dicamba	LD50 2,000 mg/kg	(DOE 1983)	(WSSA 1983)		
2,4-D	Acute Oral	2,4-D	MCPP	.99%	
MCPP	LD50 5,000 mg/kg	LD50 532 mg/kg	LD50 1,060 mg/kg		
			(WSSA 1983)		

Table 5-16 (continued).

Combination	Test	Level in Comp. 1	Level in Comp. 2	Level in Comp. 3	Level in Comp. 4
2,4-D 46.7%	Acute Oral LD ₅₀ 887 mg/kg	2,4-D 46.7% LD ₅₀ 532 mg/kg	2,4-DP 45.9% LD ₅₀ 532 mg/kg		
2,4-DP 45.9%					
2,4-D 46.7%	Acute Dermal LD ₅₀ 2,405 mg/kg	2,4-D 46.7% LD ₅₀ 1,400 mg/kg (DOE 1983)	2,4-DP 45.9% LD ₅₀ 2,000 mg/kg		
2,4-DP 45.9%					
Dicloram 17.1%	Acute Oral LD ₅₀ 2,991 mg/kg ^(m) 3,011 mg/kg ^(f)	Dicloram 17.1% LD ₅₀ 8,200 mg/kg	Triclopyr 32.5% LD ₅₀ 630-739 mg/kg		
Triclopyr 32.5%					
Dicloram 17.1%	Acute Dermal LD ₅₀ 1,485 mg/kg	Dicloram 17.1% LD ₅₀ 4,000 mg/kg	Triclopyr 32.5% LD ₅₀ 2,000 mg/kg		
Triclopyr 32.5%					
Simazine, same status listed under dicamba princep WP. acute dermal and acute oral					

a given year, the Forest Service and BLM may treat up to 156 mi² (100,000 acres) with herbicides for vegetation management. The treated area would thus comprise less than one-thousandth (less than 0.1 percent) of the total land area of the two States. Moreover, the treatments would occur for the most part in the remote areas of these either densely forested or range lands. In general, treatment units are sprayed only once in a given year, then not treated again until a number of years later. The later treatment also may be with a different herbicide.

No one individual member of the public is likely to receive repeated exposures to any of the herbicides because of the remoteness of most treatment units, the widely spaced timing of repeated treatments, and the use of a variety of herbicides for different purposes. In addition, the precautions taken by the Forest Service and BLM in their treatment operations make any dose at all to the public quite unlikely. This risk assessment used the lowest NOEL's found in chronic animal laboratory studies for comparison with estimated human doses. The risk analysis results showed that, except for amitrole and triclopyr, margins of safety for the public from realistic treatment scenarios are greater than 200. Thus, members of the public could receive doses of these herbicides repeatedly over the years, even though the chance of receiving multiple doses is negligible, and still not suffer toxic effects. Some individuals who may be particularly sensitive to amitrole or triclopyr may experience ill effects but, again, this should occur only in the unusual circumstance of repeated doses. The public can be exposed to a wide variety of other chemical compounds through both voluntary and involuntary routes of exposure. BLM acknowledges that the potentially exposed public from proposed vegetation management program do not live in a chemical free environment. However,, because of the reasons stated above additional risk to humans from Forest Service and BLM operations from year to year would be insignificant. Sensitive individuals are discussed in the next section.

Cumulative effects on workers have been considered throughout this analysis. The risk of workers experiencing toxic effects, including cancer, assumes that they are chronically exposed to these herbicides. Backpack applicators are at greatest risk from cumulative effects. Contract employees are not expected to be at any greater risk than government employees.

Factors Affecting the Sensitivity of Individuals. Individuals typically display a range of susceptibilities to toxic effects of chemicals. Factors that may affect susceptibility include diet, age, heredity, preexisting diseases, and life style (Calabrese 1978). These factors have been studied in detail for very few cases, and their significance in controlling toxicity of the proposed herbicides is not known. However, enough data has been collected on other chemicals to show that these factors can be important.

Populations at Risk

The populations at risk in herbicide spraying operations in the Pacific Northwest fall into three categories: (1) workers involved in the spray operations, (2) forest users such as hikers, hunters, and fishermen, and (3) residents of dwellings in and near the forest.

The number of workers involved in spraying operations for a typical spray year for the Forest Service and BLM is discussed in Section 2. The number of forest visitors to Forest Service and BLM land is recorded as visitor days by the agencies. The Forest Service in Region 6 averages approximately 30 million total visitor days annually. Total visitor days for BLM averages about 2 million annually in western Oregon. The number of residents living within a specified distance of Forest Service and BLM land is as follows:

<u>Distance</u>	<u>Number of Residents</u>	
	<u>Forest Service land</u>	<u>BLM land</u>
1/4 mile	29,831	30,357
1/2 mile	50,919	53,395

Again, because of the remote locations of most herbicide application sites, no member of the public should be exposed in the vast majority of operations. Silvicultural operations present the least probability of exposure, while right-of-way and facilities maintenance operations present the greatest probability of exposure.

The only possibility for exposure beyond one-half mile distance is in the extremely unlikely event of an accidental worst case spill. BLM estimates that the number of people living within a mile of its land in western Oregon is approximately 130,000. The Forest Service estimates that the number of residents with a mile of its land is approximately 100,000.

Appendix D
Human Health Risk
Assessment
(Quantitative)

Section 6

Section 6

LIST OF PREPARERS

This risk assessment was prepared by an interdisciplinary team of environmental scientists, ecosystem modelers, and toxicologists. Members of the Forest Service and BLM listed here provided the background information on vegetation management operations and herbicide use in Washington and Oregon and were the principal reviewers at each stage of its preparation. Many other members of the Forest Service and BLM not listed here participated in the review process.

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Appendix D
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References

REFERENCES CITED IN APPENDIX D

All documents referenced in this Appendix are available at universities, at libraries, or from Federal agencies such as the Forest Service or the U.S. Environmental Protection Agency (EPA). All EPA documents can be obtained through requests to EPA's Freedom of Information Office, Washington, D.C. 20460.

In the text of this document references are cited in parentheses using the author-year system of citation. When an organization (such as a Government agency or scientific society) is listed as the author in the parenthetical citation, an acronym or an abbreviated form of that organization's name generally is used in place of its full title. Below is a list of acronyms and abbreviations that are used in citations, along with the corresponding full titles that are used in this reference section.

BLM	U.S. Department of the Interior, Bureau of Land Management
DOE	U.S. Department of Energy
EPA	U.S. Environmental Protection Agency
NCI	National Cancer Institute
OSTP	U.S. Office of Science and Technology Policy
USDA	U.S. Department of Agriculture
WSSA	Weed Science Society of America

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Appendix D
Human Health Risk
Assessment
(Quantitative)

Glossary

GLOSSARY

A

Acceptable Daily Intake (ADI). The maximum dose of a substance that is anticipated to be without lifetime risk to humans when taken daily.

Acetone. A colorless, volatile liquid that is useful as a solvent. It is found in the blood and urine when fats are not properly metabolized.

Acid Equivalent (a.e.). The amount of active ingredient expressed in terms of the parent acid.

Active Ingredient (a.i.). The chemical in a herbicide that is primarily responsible for its phytotoxic or herbicidal action.

Acute Toxicity. The quality or potential of a substance to cause injury or illness shortly after exposure to a relatively large dose.

Adenoma. An abnormal growth of glandular tissue.

Adenocarcinomatous. Referring to a malignant (cancerous) adenoma.

Adsorption. Adhesion of substances to the surfaces of solids or liquids. Technically, the attraction of ions of compounds to the surfaces of solids or liquids.

Ames Assay. A type of short-term test using bacteria in laboratory cultures to assess the mutagenic potential of a substance.

Assay. A test or measurement used to evaluate a characteristic of a chemical. See Bioassay.

Atopic. Displaced or not located in the usual position.

B

Bacteriophage. A group of transmissible agents (bacterial viruses) capable of destroying certain bacterial cells.

Bile Ducts. Passages that convey the bile from the liver and gall bladder to the small intestine.

Bioaccumulation. The process of a plant or animal selectively taking in or storing a persistent substance. Over a period of time, a higher concentration of the substance is found in the organism than in the organism's environment.

Bioassay. A method for quantitatively determining the concentration of a substance by its effect on a suitable animal, plant, or microorganism under controlled conditions.

Boom (herbicide spray). A tubular metal device that conducts an herbicide mixture from a tank to a series of spray nozzles. It may be mounted beneath a helicopter or a fixed-wing aircraft or behind a tractor.

Buffer Strip/Zone. A strip of vegetation that is left or managed to reduce the impact that a treatment or action on one area would have on another area.

C

Carcinogenic. Capable of producing or inciting cancer.

Carcinoma. A malignant or cancerous tumor.

Chemical Degradation. The breakdown of a chemical substance into simpler components through chemical reactions.

Cholangiofibrosis. An abnormal formation of fibrous tissue within the bile duct of the gall bladder.

Chromosome. Microscopic structures within the cell that are composed of DNA and the genes (hereditary determiners).

Chronic (effects or toxicity). Having poisonous or deleterious effects from prolonged exposure or repeated administration of a chemical.

Conifer. An order of the Gymnospermae, comprising a wide range of trees, mostly evergreens that bear cones and have needle-shaped or scalelike leaves; timber commercially identified as softwood.

Crossing Over. The breaking and exchanging of parts of chromosomes between chromosome pairs during cell division.

Cytogenetic. Refers to the structure or function of chromosomes within cells.

D

Degradation. See chemical degradation.

Dermal Exposure. That portion of an amount of toxic substance that an organism receives as a result of the substance coming into contact with the organism's body surface.

Dislodgeable Residue. A pesticide residue that can be removed from surfaces such as foliage by physical contact.

DNA. Deoxyribonucleic acid. Any of various nucleic acids that are the molecular basis of heredity in many organisms.

Dominant Lethal Assay. A toxicity test whereby a male animal (usually a rodent) is exposed to a chemical substance and later sequentially mated with two female animals. The females are sacrificed, and the number and status of the fetuses is recorded.

Dose. The amount of chemical administered or received by an organism, generally at a given point in time.

Drift. That portion of a sprayed chemical that is moved by wind off a target site.

E

Ecosystem. An interacting system of organisms considered together with their environment; for example, marsh, watershed, and lake ecosystems.

Environmental Impact Statement (EIS). A formal document to be filed with the Environmental Protection Agency that considers significant environmental impacts expected from implementation of a major Federal action.

E. coli or Escherichia coli. A common species of bacteria used in many areas of biological research, including mutagenicity testing.

Ester. A compound formed by the reaction of an acid and an alcohol, generally accompanied by the elimination of water.

Exposure Analysis. The estimation of the amount of chemical that is in an organism's environment and available for uptake into the body.

F

F₀. In genetics and reproduction studies, it pertains to the first parents' generation.

F₁. In genetics, it refers to the first generation of offspring from the F₀ generation.

Fate. The course of an herbicide in an ecosystem or biological system after it has been applied; including metabolism, microbial degradation, leaching, and photodecomposition.

Fetotoxic. Capable of producing adverse effects in a developing fetus.

Fibroblast. Any cell from which connective tissue is developed.

Formulation. A chemical mixture that includes a certain percentage of active ingredient (technical chemical) with an inert carrier.

G

Gavage. Feeding by way of a tube inserted into the stomach.

Gene. The basic unit of heredity. Each gene occupies a specific place (locus) on a chromosome.

Genotoxic. Harmful to genetic material (DNA).

Germ Cell. A functional sex cell that combines with the opposite sex cell for fertilization, for example, sperm, egg.

Global 82. A computer program by Howe and Crump (1982) used to fit the multistage or one-hit models to experimental cancer data.

H

Half-Life. The amount of time required for half of a compound to degrade.

Hazard Analysis. The determination of whether a particular chemical is or is not causally linked to particular harmful effects.

HDT. Highest dose tested.

Hectare (ha). 10,000 square meters, or approximately 2.47 acres.

HeLa Cell Line. A human cell line originally derived from cancerous breast cells.

Hematocrit. The percentage by volume of red blood cells in a given volume of blood.

Hemoglobin. The iron-containing compound in red blood cells that functions to carry oxygen from the lungs to the tissues.

Hepatoma. A tumor of the liver.

Herbaceous. A plant that does not develop persistent woody tissue above the ground.

Herbicide. A chemical used to control, suppress, or kill plants, or to severely interrupt their normal growth processes.

Heritable. Capable of being passed on from parents to offspring.

Histology. The study of the microscopic structure of tissue.

Histopathologic. Referring to tissue changes characteristic of disease.

Hydrolysis. Decomposition or alteration of a chemical substance by water.

Hyperplasia. An excessive proliferation of normal cells in the tissue of an organ.

Hypertrophy. An increase in size of an organ or structure that does not involve tumor formation.

Hypohatchet. A tool used to inject herbicide into a tree trunk or woody stem.

I

In Vitro. Pertaining to a test that is conducted outside the living body and in an artificial environment such as a test tube or petri dish.

In Vivo. Pertaining to a test that is performed within the living body of the organism.

Intraperitoneal. Related to a structure or process occurring within the peritoneum, a membranous lining of the body cavity.

Intravenous. Within or into a vein.

K

Kilogram (kg). One thousand grams; or approximately 2.2 pounds.

L

Label. All printed material on or attached to a pesticide container as required by law.

Latency Period. The time between a stimulus and its response.

LC50. A lethal concentration rate at which 50 percent of the test animals will be killed. It is usually used in the testing of fish or other aquatic animals.

LD50. The dosage of toxicant, expressed in milligrams of toxicant per kilogram of animal body weight, required to kill 50 percent of the animals in a test population when given orally.

LDT. Lowest dose tested.

Leach. Usually refers to the movement of chemicals through soil by water; may also refer to the movement of herbicides out of leaves, stems, or roots into the air or soil.

Least Squares Estimation. A mathematical approach used to fit a straight line (or other models) so that the sum of the squares of the vertical distances of the data points from the line will be a minimum.

Lowest Effect Level (LEL). The lowest dose tested that results in an effect in a test organism.

Linear Regression. A mathematical procedure used to draw a straight line that best fits a set of data points on a graph.

Log-Probit Model. An equation used to describe the relationship between dose and the probability of contracting cancer. This equation can be derived by assuming that humans (or animals) have various susceptibilities, but that at very low doses none has a significant risk.

Lymphocyte. A cell of the lymphatic system, or a special type of white blood cell.

Lymphoma. A general term for the growth of new tissue in the lymphatic system.

M

Malignant. Used in reference to a tumor; indicating the presence of cancer and tending to grow worse and spread within an organism.

Margin of Safety (MOS). The ratio between the no-observed-effect level (NOEL) and the estimated dose.

Metabolism. The chemical changes in living cells by which energy is provided for vital processes and new material is assimilated.

Metabolite. A product of the chemical changes in living cells that provide energy and assimilate new material.

Microbial Degradation. The breakdown of a chemical substance into simpler components by bacteria or other microorganisms.

Microgram (ug). One millionth of a gram.

Mitigation Measures. Means taken to avoid, compensate for, rectify, or reduce the potential adverse impacts of a proposed action.

Mitotic. Pertaining to the process of cell division that results in two cells having the same number of chromosomes as the original cell.

Multistage Model. An equation used to describe the relationship between dose and the probability of contracting cancer. This equation, commonly used by EPA, assumes that several successive events must occur to produce cancer.

Mutagen. A substance that tends to increase the frequency or extent of genetic mutations (changes in hereditary material).

Mutagenic. Capable of producing genetic defects in an organism.

N

Necrosis. Death of a cell or group of cells as a result of injury, disease, or other pathologic state.

Neoplastic. Pertaining to new abnormal tissue formation (neoplasms).

Neuropathy. Any disease affecting neurons, the fundamental functional units of nervous tissues.

NOEL (no-observed-effect level). The dose level at which no toxic effects are observed in a test organism.

Noxious Weed. A plant regulated or identified by law as being undesirable, troublesome, and difficult to control.

Nucleic Acid. A group of complex molecules found in cells, composed of phosphoric acid, sugars, and nitrogen bases. Includes DNA and RNA.

O

ODT. Only dose tested.

Omphalocele. A congenital hernia of the navel.

Oncogenic. Capable of producing or inducing tumors in animals, either benign (noncancerous) or malignant (cancerous).

Oncology. The branch of medicine which studies tumors.

One-Hit Model. An equation used to describe the relationship between dose and the probability of contracting cancer. This equation, used at one time by EPA, predicts the greatest cancer probability at low doses of all commonly used models.

Organic Material. An accumulation of decayed and resynthesized plant and animal residues with a high capacity for holding water and nutrients.

Ossification. The formation of bone.

P

Papillary. Resembling or composed of small protuberances or elevations.

Parenteral. Injection of a substance into the body through any route other than the digestive tract.

Particulates. Finely divided solid or liquid particles in the air or in an emission; includes dust, smoke fumes, mist, spray, and fog.

Pathology. The study of the nature and cause of disease with respect to functional and structural changes.

Persistence. The resistance of a pesticide to metabolism and environmental degradation.

Pesticide. As defined by FIFRA, any substance or mixture of substances intended for preventing, destroying, repelling, or mitigating any pest, and any substance or mixture of substances intended for use as a plant regulator, defoliant, or desiccant.

Photochemically Reactive. A property of substances or particles whose structures may be changed when solar energy is absorbed.

Photolysis (photodecomposition). The breakdown of a substance, especially a chemical compound, into simpler components by the action of radiant energy such as sunlight.

Photosynthesis. Formation of carbohydrates in the tissues of plants exposed to light.

Phytotoxic. Injurious or lethal to plants.

Pituitary Gland. A small, oval endocrine gland attached by a stalk to the base of the brain and consisting of an anterior and a posterior lobe; it secretes hormones that influence body growth, metabolism, and so forth; hypophysis.

ppm (parts per million). A unit for measuring the concentration of a substance, such as a pesticide, in a carrier medium, such as food or water. For example, where the concentration is 1 ppm, the weight of the substance is 1 millionth the weight of the carrier medium; thus, 1 ppm is equal to 1 milligram of substance per kilogram of food or organism body weight, and it is equal to 1 milligram of substance per liter of water.

Proliferation. The rapid and repeated reproduction of new cells.

Pulmonary. Concerning or involving the lungs.

Pyrolysis. Chemical breakdown caused in the process of combustion.

R

Recreation Visitor Day (RVD). Twelve visitor hours, which may be aggregated continuously, intermittently, or simultaneously for one or more persons.

Reentry. The return of a worker to an area that has recently been treated with a pesticide.

Renal Tubule. The functional unit of the kidney where urine is formed; nephron.

Residue. The quantity of a herbicide or its metabolites remaining in or on soil, water, plants, animals, or surfaces.

Resorption. Act of removal by absorption.

Risk Analysis. The description of the nature and often the magnitude of risk to organisms, including attendant uncertainty.

Runoff. That part of precipitation, as well as any other flow contributions, that appears in surface streams, either perennial or intermittent.

S

Safety Factor. A factor conventionally used to extrapolate human tolerances for chemical agents from no-observed-effect levels in animal test data.

Salmonella. A genus of bacteria used in mutagenicity testing.

Sediment. Organic matter or soil that settles to the bottom of a liquid.

Shrub. A plant with persistent woody stems and relatively low growth form; usually produces several basal shoots as opposed to a single bole; differs from a tree by its low stature and nonarborescent form.

Silviculture. The branch of forestry dealing with the care, development, and reproduction of forest trees or stands of timber.

Sister Chromatid Exchange (SCE). A short-term test conducted with laboratory cell cultures to assess the genetic damage caused by a chemical or physical influence.

Spot Treatment. Application of a herbicide to a small selected area as opposed to broadcast application.

Subchronic. The effects observed from doses that are of intermediate duration, usually 3 months (90 days).

Subcutaneous. Beneath the skin, or to be introduced beneath the skin.

Surfactant. A material that improves the emulsifying, dispersing, spreading, wetting, or other surface-modifying properties of liquids.

Systemic Herbicide. An herbicide that is moved within the plant. In a more restricted sense, refers to herbicides that are applied to the foliage and move downward through the living tissue to underground parts.

Systemic Toxicity. Effects produced as a result of the distribution of a poison or foreign substance from the point of exposure to a distant site within the body.

T

T₃. Triiodothyronine. A chemical measured in tests which evaluate the functioning of the thyroid gland.

T₄. Tetraiodothyronine. A chemical measured in tests which evaluate the functioning of the thyroid gland.

Teratogen. A substance tending to cause developmental malformations in unborn human or animal offspring.

Teratogenesis. The development of abnormal structures in an embryo.

Teratogenic. Capable of producing or inciting the development of malformations in an embryo.

Teratology. The study of malformations in organisms.

Thiourea. A colorless crystalline form of urea containing sulfur in place of oxygen.

Thymus. A relatively small organ located in the upper chest that is important in the development of the immune system in newborn and young animals.

Thyroid Gland. A large, ductless gland lying in front of and on either side of the trachea and secreting thyroxine which regulates the growth of the body.

Thyroid Stimulating Hormone (TSH). A chemical secreted by the pituitary gland intended to cause the thyroid gland to produce its hormones.

Toxicity. A characteristic of a substance that makes it poisonous.

Toxicology. The science dealing with the study of the adverse biological effects of chemicals.

Tumor. A new growth of tissue that forms an abnormal mass and performs no physiologic function. It usually develops independent of and unrestrained by the normal principles of biological growth.

Tumorigenesis. The formation and/or development of a tumor (oncogenesis).

V

Volatility. The quality of evaporating readily at normal temperatures and pressures.

Volatilization. The vaporizing or evaporating of a chemical substance.

W

Wettable Powder (WP). A finely divided dry formulation that can be readily suspended in water.

Appendix D
Human Health Risk
Assessment
(Quantitative)

Attachment A

Attachment A

DETAILS OF MUTAGENICITY AND CARCINOGENICITY TESTING

MUTAGENICITY

This subsection presents the details of mutagenicity tests conducted on amitrole, atrazine, bromacil, 2,4-DP, and dicamba.

Amitrole

The mutagenic potential of amitrole is summarized by the following information from EPA (1985a):

Amitrole has been evaluated in a variety of mutagenicity test systems. Although positive results were reported by Braun et al., 1977, (using added nitrite) in Salmonella and by Venitt and Crofton-Sleigh (1981) in Salmonella and E. coli, 49 other Salmonella gene mutation tests and 9 other E. coli tests were negative. The validity of the two positive studies is questionable. The weakly positive results by Carere et al. (1976, 1978, and 1981) were in an unvalidated system using unusual bacteria. The mechanisms for these positive results reported for the DNA repair assays cannot be determined without positive gene mutation or chromosome aberration assays. The negative results in the sister chromatid exchange assay in mammalian cells in culture (which is a very sensitive assay) and the chromosome aberration assays in cultured human lymphocytes or in vivo mouse bone marrow cells cast doubt on the significance of the DNA repair assays. Amitrole does not present a potential for heritable genetic effects.

Amitrole is able to induce transformation of cultured cells. It was positive in four in vitro transformation studies using rat and hamster cells (Pienta, 1977; Inoue, 1981; Dunkel, 1981; Styles, 1981) following treatment of 0.1 to 100 ug/ml. This test is used to establish the malignant activities of test compounds on mammalian cells in vitro. Cells treated in vitro with chemical carcinogens give rise to foci of cellular growth superimposed on the cell monolayer. If these foci are picked from the cultures, grown to larger numbers, and injected into animals, a malignant tumor will be obtained, in most cases. Therefore, the appearance of piled-up colonies in treated cell cultures is correlated with malignant transformation. In addition, weak cellular transformation capacity was observed in EUE cells (no data presented, only summary) (Benigni, 1980).

These transformation assays are not able to determine a mechanism for tumor formation and do not necessarily show that a transformation inducer is genotoxic. These results support oncogenicity potential but not necessarily mutagenicity potential.

Atrazine

Atrazine did not induce mutations in microbial assay systems evaluated by EPA (1984c). All of the following positive results were reported in USDA (1984). Atrazine represents an unusual situation because many of the positive genotoxicity studies were conducted using a metabolic activation system of plant origin. Atrazine alone or when tested in vitro in tests using animal metabolic systems was not genotoxic in most cases. Because plant metabolism is not generally considered important in developing a human hazard assessment, the relevance of the "plant-activated" tests in a risk analysis is doubtful.

Atrazine is not genotoxic in bacteria or yeast directly or with the typical rodent S9 activation. Yeast tests for mitotic crossing over and mutation do show positive responses, but only when tested with atrazine incubated with plant cell extracts. Bacteriophage T4 mutation and a B. subtilis test for repairable DNA damage were negative with atrazine alone. A Streptomyces coelicolor mutation assay was positive, but this assay appears to have very little reliability to discriminate true positives and false positives.

In vitro tests with mammalian cells also fail to respond to atrazine directly or with mammalian metabolism (aberrations and sister chromatid exchange tests in hamster cells were negative), but when hamster V79 cells were exposed to atrazine in the presence of plant cell extracts, the chemical was reported to be mutagenic. Positive unscheduled DNA synthesis (UDS) effects in EUE cells were also reported with atrazine plus plant cell extracts.

Atrazine was reported to be genotoxic directly in plants (mutations at the waxy locus in corn; chromosome aberrations in plant cells) and the mold Aspergillus nidulans (crossing over). Mutation studies in Aspergillus were conducted with plant cell extracts. In the presence of plant cell extracts, a mutation to 8 azaguanine resistance was reported for Aspergillus.

Atrazine was reported to induce sex-linked recessive lethal mutations in the fruit fly (Drosophila melanogaster) in one of two studies.

In vivo studies measuring chromosome aberrations in rodent bone marrow and dominant lethal mutations in the mouse were reported positive at dose levels of 2,000 and 1,500 mg/kg, respectively.

Positive responses in the Drosophila sex-linked recessive lethal assay (which measured gene mutations in germ cells) and the mouse dominant lethal assay (which measured chromosome alterations in germ cells) both indicate a potential for mutagenic hazard. Bone marrow chromosome aberration effects support this conclusion. The in vivo responses were observed only at very high levels of atrazine equal to or exceeding 1.5 gm/kg.

Bromacil

The mutagenicity studies submitted to EPA for the registration of bromacil were deemed unacceptable; therefore, no validated mutagenicity studies have been completed (EPA, 1984d).

Because of the potent mutagenicity of 5-bromouracil, which is a structurally related chemical, the metabolic fate of bromacil has been examined to determine whether the formation of 5-bromouracil occurs in vivo. Metabolic fate studies indicate that 5-bromouracil was not isolated from the urine and feces of rats exposed to bromacil or from the urine of bromacil production plant workers (DOE, 1983).

2,4-DP

A very limited assessment of the genotoxicity of 2,4-DP is available. 2,4-DP was nonmutagenic when tested in three nonactivated microbial assays (EPA, 1984f). 2,4-DP was not mutagenic to Salmonella in the Ames test. A sample was tested for induction of repairable DNA damage in E. coli. The results were positive with metabolic activation. Positive results were obtained when 2,4-DP was tested in the nonactivated Saccharomyces cerevisiae reverse mutation assay, the Saccharomyces cerevisiae mitotic gene conversion assay, and the activated E. coli assay for unscheduled DNA synthesis (EPA, 1984f).

A single in vivo micronucleus study in mice was conducted, but results were not reported because the study was concluded to be invalid by EPA.

Dicamba

Dicamba was nonmutagenic when tested in various microbial assays for gene mutations and a dominant lethal assay with mice (USDA, 1984). Positive mutagenic responses were observed when dicamba was tested in the B. subtilis and E. coli toxicity assays for primary DNA damage (USDA, 1984); however, these tests are not useful in determining human mutagenic potential.

CARCINOGENICITY

This subsection presents the details of oncogenicity tests conducted on amitrole, asulam, atrazine, bromacil, 2,4-D, 2,4-DP, glyphosate, picloram, and simazine.

Amitrole

Amitrole has been shown to induce a high incidence of thyroid tumors in laboratory animals. Amitrole also induces liver tumors in mice when fed at levels in excess of the maximum tolerated dose; it also produces thyroid carcinomas along with the liver tumors (EPA, 1984a; EPA, 1985a).

Amitrole is an antithyroid compound that resembles thiourea in its effect on the thyroid gland. Prolonged administration leads to a reduction in the levels of circulating thyroid hormones and a compensatory increase in the production of thyroid stimulating hormone (TSH). This results in enlargement of the thyroid (goiter) and in adenomatous (tumor growth of the glandular epithelium) changes.

There has been some disagreement among pathologists regarding the nature of the changes that have been induced in the thyroid glands of treated laboratory animals in some experiments. Some pathologists interpreted some of the

changes to be cancerous, while others judged them to be nonmalignant (e.g., Jukes and Shaffer, 1960). In a 2-year rat feeding study conducted by Hazelton Laboratories, Inc., rats fed 100 ppm of amitrole had greater than 50 percent (15 of 27) incidence of thyroid adenoma. In rats fed 10 and 50 ppm, the incidence was lower (1 of 27 and 3 of 27, respectively), and there were no adenomas in the control group (Hodge et al., 1966; Jukes and Shaffer, 1960). In another 2-year rat feeding study by Bayer (1979, cited in EPA, 1985a), significant increases in thyroid tumors were observed at 100 ppm. Also, when rats were pulse-fed amitrole alternating with a control diet in an effort to minimize thyroid stimulation, significant increases in thyroid tumors were observed at the higher dose levels (Food and Drug Research Lab, 1981, cited in EPA, 1985a).

In another experiment, rats were fed 2,500 ppm in their drinking water for 70 weeks (Tsuda et al., 1976). Of 26 surviving rats, 100 percent had goiter development, and 73 percent had invasive thyroid lesions, and 11.5 percent had papillary adenoma. In two rats, liver damage occurred because of irregular proliferation of the bile ducts. Two other rat feeding studies and one hamster study found no increase in thyroid tumor incidence (EPA, 1985a).

Most exposures to workers and the public can be expected to be dermal, and dermal exposure in a mouse study did not yield similar effects. In a long-term (575 days) mouse study, subcutaneous injections (10 mg/mouse) and weekly skin applications in acetone produced no tumors (Hodge et al., 1966).

Other studies have shown that amitrole can produce liver tumors. For example, Feinstein et al. (1978) induced liver tumors by feeding high levels of amitrole to mice. When mice were fed 1 percent amitrole in their diets for 1 year, during 4 out of each 5 weeks, 100 percent of them developed liver tumors by 1 year after weaning. Only a few mice had thyroid tumors. However, this strain of mice (C3H) has a high natural incidence of liver tumors, and the concentration of amitrole given was so high that survival was affected for other reasons. Innes et al. (1969) also reported the induction of liver tumors in mice by amitrole. However, in this study, the doses also were so high (2,192 ppm) that survival was affected. Several other studies have reported no increase in the incidence of liver tumors (EPA, 1985a).

In some studies, amitrole has also been shown to cause pituitary tumors. In a 2-year rat study, pituitary tumors were increased at 100 ppm in females (Bayer, 1979, reported in EPA, 1985a). In another 2-year study, female rats had a significant incidence of pituitary tumors at the highest dose tested (100 ppm), whether it was administered continuously or intermittently (Food Drug Research Lab., 1981, reported in EPA, 1985a).

EPA has classified amitrole in Category B₂, indicating that it is a probable human carcinogen (EPA, 1985a). EPA has also stated in its Amitrole Toxicology Summary that the scientific evidence "strongly supports the use of a threshold approach in assessing the oncogenic risk of amitrole, realizing, however, that this is a radical departure from previously used risk assessments" (EPA, 1985a). EPA also concluded that amitrole is not mutagenic, and animal data suggest a "secondary carcinogenic" effect due to

increased production of thyroid-stimulating hormone (EPA, 1985a). Consequently, EPA has suggested the use of a log-probit model in carcinogenic risk estimation for thyroid and pituitary tumors to better represent the threshold effect (EPA, 1985a). However, EPA has also used the multistage model in its risk assessment on thyroid tumors for comparison with the log-probit model predictions and for the assessment of risk from liver tumors (EPA, 1985a). One report not considered by EPA has suggested that amitrole may act via a mutation mechanism, indicating that caution may be warranted in low-dose extrapolation (Tsutsui et al., 1984).

Asulam

Asulam was oncogenic in one of three studies conducted. Two studies with mice resulted in no oncogenic effects after 18 months at the highest dose tested of 5,000 ppm (750 mg/kg/day).

A 107-week feeding study with rats resulted in a statistically significant increase in carcinoma of thyroid c cells at 1,000 ppm (20 mg/kg/day) in males. A significant increase in adrenal medullary hyperplasia was observed in males at 25,000 ppm (EPA, 1985d; EPA, 1983).

Atrazine

In an 18-month mouse feeding study, atrazine did not induce any tumors at 12.5 mg/kg/day (Innes et al., 1969); however, oncogenic effects were observed in a 2-year rat feeding oncogenicity study. The following is a summary of atrazine oncogenicity information from EPA (1986):

Study Results: Technical grade atrazine was evaluated for chronic oral toxicity and oncogenicity in Sprague-Dawley rats fed dietary concentrations of 0 (untreated control), 10, 70, 500 and 1,000 parts per million (ppm) for 2 years. A number of chronic toxicity symptoms were observed: reduced body weight for animals in the 500 and 1,000 ppm test groups, reduced red cell parameters (such as red blood cell count and hemoglobin) for females in the 1,000 ppm test group, and decreased glucose levels in animals fed 1,000 ppm. These data indicate a systemic toxicity NOEL of 70 ppm.

There was a significant increase in total mammary tumors in female rats fed 70, 500, and 1,000 ppm atrazine. These tumors were observed as tissue masses or lumps even before the animals were sacrificed for pathologic examination.

Preliminary studies conducted to evaluate the carcinogenic potential of Fogard S^R (25 percent atrazine and 37.5 percent simazine) resulted in the formation of malignant lymphomas in mice after exposure to doses equivalent to 0.104 mg/kg of atrazine for a 7-month test period (Donna et al., 1981). The doses were given by subcutaneous and intraperitoneal injection. Tumor formation occurred as a result of both types of injection. The numbers of test animals were relatively small. Of the 24 mice receiving subcutaneous injections, 4 developed tumors, and of the 20 mice receiving intraperitoneal injections, 2 developed tumors. It is possible that the tumor formation observed in this study may be a function of the route of administration or a result of other chemicals in the formulation because atrazine and simazine have not been shown to be carcinogenic in other chronic studies, even at much higher doses.

N-Nitrosoatrazine

N-Nitroso derivatives of atrazine are carcinogenic to laboratory animals and mutagenic (Krull et al., 1980). Concerns have been raised over the potential for the nitrosation of atrazine to N-Nitrosoatrazine (NNA) under field conditions and over the potential toxicity of this compound.

Kearney et al. (1977) examined the formation and degradation of N-Nitrosoatrazine in soils and aquatic environments. Their results indicate that the formation of NNA is highly unlikely under normal application rates of atrazine (2 ppm) in agricultural soils of pH 5 to 7. They used elevated levels of nitrogen fertilizer (approximately 100 ppm) that are much higher than those used in forestry. Most of the NNA added to soil was converted relatively quickly to atrazine by denitrosation.

The degradation of NNA in aquatic environments is very rapid primarily because of photolysis (Wolfe et al., 1976).

Bromacil

Bromacil was oncogenic in one of two studies conducted. No oncogenic effects were observed in rats at doses up to 1,250 ppm (62.5 mg/kg/day) and 2 years of exposure. Liver tumors (hepatocellular adenomas) were observed in an 18-month study with mice at doses of 5,000 ppm (750 mg/kg/day) (EPA, 1985e).

2,4-D

A number of studies have assessed the carcinogenicity of 2,4-D, and thus far, there are no conclusive data demonstrating the carcinogenicity of 2,4-D (International Agency for Research on Cancer, 1977; Mullison, 1981; State of Minnesota, 1978, all as cited in USDA, 1984). However, there is also general agreement that none of these studies were adequate (EPA, 1982a; International Agency for Research on Cancer, 1977, as cited in USDA, 1984; WHO, 1984). At least one scientist, Dr. M. Rueber, disputes the conclusion that a carcinogenic effect of 2,4-D has not been shown (Rueber, 1979, as cited in BLM, 1985).

EPA has recently reviewed a long term study on the oncogenic potential of 2,4-D. Preliminary findings indicate an increased incidence of brain tumors in rats. But EPA's review of the recent cancer study is not yet complete. EPA has requested an independent expert to review the brain tissue slides from this study and may also request a review of this study by the Scientific Advisory Panel. Thus a thorough review of this study may take months to complete. Therefore, EPA does not believe it is now appropriate to derive a specific numerical estimate of cancer potency based on the new data, but has stated, that from its preliminary review the level of cancer potency indicated by the reported results would be of about the same order of magnitude as the potency value based on the Hansen study that has been used in previous risk analyses (EPA, 1986c).

At 106 weeks, a preliminary pathology report from a recent mouse study found that 2,4-D was not oncogenic at dosages of 1, 15, and 45 mg/kg/day (Hazelton Laboratories, 1986).

Recently, Hoar et al. (1986) completed a case control epidemiologic study in Kansas, examining the risk of lymphoma and soft-tissue sarcoma in men from agricultural herbicide exposure. The study found no association between exposure and soft-tissue sarcoma or Hodgkin's disease, but observed a significant association for non-Hodgkin's lymphoma and phenoxyacetic acid herbicide exposure, especially 2,4-dichlorophenoxyacetic acid exposure. In addition, individuals exposed to herbicides for more than 20 days per year had a sixfold increase in non-Hodgkin's lymphoma. This study, however, suffers from the same inherent limitations as other case-control studies, mainly that it relies on the subject's and the next of kin's recall of exposure status. If recall is faulty, then misclassification occurs. Assessing exposure-disease relationships in these types of epidemiological studies is especially difficult (Thomas, 1986). For example, common exposures to other carcinogenic agents or other factors may result in disease but be undiscovered in the interview and confound the results. Thus, uncontrolled confounding factors in observational epidemiological studies can be particularly troublesome in interpreting the results. The apparent dose-response relationship observed in the Hoar et al. (1986) study for non-Hodgkin's lymphoma (NHL) is of public health concern and needs further examination.

A recent review of the Hoar et al. (1986) study conducted for EPA by Brian MacMahon, M.D., Ph.D. of the Harvard School of Public Health, concluded:

In my opinion the weight of evidence does not support the conclusion that there is an association between exposure to 2,4-D and NHL. It is axiomatic that, except when relative risks are very high--and sometimes even then--no single study will establish an association between an exposure and an outcome. The acceptance of an association depends on a number of studies showing consistent results across populations and across different epidemiologic methods. The study of Hoar et al. is a strong study--strong enough on its own to establish a hypothesis of relationship of exposure to 2,4-D with some small proportion of cases of NHL--a hypothesis that clearly deserves attempts at refutation or support in other populations. When one attempts to place the results of this study among the results of those published previously, the picture becomes very confusing--much more so than if Hoar et al. had been the only study published. Taken as a whole, I believe that the weight of evidence indicates that an association between 2,4-D and NHL remains a hypothesis that is still to be tested. I am unwilling to speculate as to whether 2,4-D causes NHL (or some cases of NHL) until the evidence is clear that there is an association between them.

Now under way are at least two more studies that should be helpful in assessing risk to humans from the use of 2,4-D and other phenoxy herbicides (Colton, 1986). In view of the uncertainty regarding the carcinogenicity of 2,4-D, a cancer risk analysis was conducted in this risk assessment under the assumption that 2,4-D is carcinogenic.

2,4-DP

Two 2,4-DP oncogenicity studies have been submitted to EPA (EPA, 1984f). One of these, an 18-month study involving Swiss-Webster CD-1 mice, was negative because the rate of incidence of the tumors that were found in the mice were unrelated to increasing doses of 2,4-DP. Some increase in the rate of hepatomas was noted in the highest dose group (300 mg/kg), but this increase was attributed to liver trauma resulting from feeding a dose greater than the maximum tolerated dose of 2,4-DP.

A 2-year oncogenicity study involved Sprague-Dawley rats fed at dose levels of 0, 50, 100, and 200 mg/kg. In this study also, the highest dose group showed signs of general toxicity because they were fed more than the maximum tolerated dose of 2,4-DP. At 60 weeks, the high-dose group was reduced to 150 mg/kg because of general toxic effects. Females at all dose levels had high rates of tumor incidence, but they did not show a dose-related response. However, males showed a significant increase in the rate of incidence of malignant tumors, with a corresponding decrease in the rate of benign tumors. The males had increased levels of both pituitary carcinomas and thyroid carcinomas. Brain tumors were also observed in one group, in both males and females, although they were determined not to be treatment related.

Glyphosate

A 26-month rat feeding study found no oncogenic effects at doses up to 31 mg/kg day (EPA, 1984k). However, this study has been downgraded to supplementary by EPA because the maximum tolerated dose (MTD) was not reached at the high dose. Benign kidney tumors (renal tubular adenomas) were found at a highest dose level (30,000 ppm) in a 2-year mouse feeding study, however, the findings were equivocal. The EPA Science Advisory Panel (SAP) has reviewed all relevant data and concluded that the oncogenic potential of glyphosate could not be determined from existing data and proposed that the study be repeated to clarify these findings (EPA, 1986c). The EPA is requiring that the mouse study be repeated with more animals in each test group to increase the statistical power of the study.

N-Nitrosoglyphosate

N-Nitroso derivatives of some herbicides are carcinogenic and mutagenic (Young and Khan, 1978; Braun et al., 1977). It has been suggested that the herbicide glyphosate may include N-Nitrosoglyphosate (NNG) as a trace contaminant, or that the compound may be formed in the environment after herbicidal application (Dost, 1983; Newton et al., 1984). However, EPA has determined that NNG does not occur as a contaminant in significant amounts in the herbicide glyphosate to pose a hazard to human health (Dost, 1983). Newton et al. (1984) found traces of NNG (approximately 0.02 ppm) in one foliage sample and one forest litter sample after aerial application of glyphosate; however, they concluded that this may have been because of the evaporation procedure used in the analysis.

Nitrosation in soil generally requires elevated nitrite levels and a pH of 3 to 4. Nitrite levels in forest area soils are generally much less than those in agricultural soils. Several studies have been conducted to measure the extent of nitrosation of glyphosate in soil with respect to temperature, pH, and organic matter content (Khan and Young, 1977; Young and Khan, 1978). NNG formed in several types of soil that were treated with glyphosate and nitrite. Levels of 5 ppm of NNG were reached when glyphosate was applied at approximately 185 ppm. This application rate is 90 to 100 times greater than normal rates. No NNG formed at glyphosate concentrations of 5 ppm and nitrite concentrations of 2 ppm. It was concluded that NNG is not likely to form in soils at the recommended application rates of 2.24 kg/ha, which is similar to application rates used in forestry.

Picloram

There is scientific controversy concerning the interpretation of studies on the potential of picloram to cause cancer. A rat oncogenicity study, during which test animals were exposed to an average of 14,875 ppm (743 mg/kg/day), was negative for oncogenic effects in males. Neoplastic nodules were observed in females at a statistically significant rate (EPA, 1984q). An oncogenic mouse study resulted in the absence of oncogenic effects at dietary exposure levels ranging from 5,000 to 15,000 ppm (750 mg/kg to 2,250 mg/kg)(EPA, 1984q and EPA, 1984m). Because there is some uncertainty regarding the potential for carcinogenicity of picloram, a worst case approach was taken to calculate cancer risks.

The Gulf Research Institute conducted a carcinogenic bioassay of picloram in rats and mice for the National Cancer Institute (1978). The mouse study produced no indication of an oncogenic response resulting from dietary exposure. Exposure consisted of as much as 5,000-plus ppm in the diet for the greatest part of their lifetime (as cited in EPA, 1984m).

The rat study, however, was negative for oncogenic effects in males, while female rats exhibited a statistically significant increase over control rats in the rate of formation of neoplastic nodules in the liver. Exposures ranged on a time-weighted average up to 14,875 ppm in the diets (743 mg/kg) (as cited in EPA, 1984m). The study concluded that the findings were "suggestive of ability of the compound to induce benign tumors in livers of female Osborne-Mendel rats."

According to a classification scheme developed by the National Cancer Institute (NCI), picloram was classified as a chemical for which evidence of carcinogenicity in animals was equivocal at best (Griesmer and Cueto, 1980, cited in BLM, 1985). However, one scientist has interpreted the data differently and has concluded that picloram was carcinogenic for all test animals except mice at the lowest dose (Rueber, 1981). This interpretation disagrees with the panel of experts who interpreted the data for the National Cancer Institute. More research on picloram's potential carcinogenicity is currently in progress at the Dow Chemical Company and is scheduled for completion in 1986. A new rodent (rat) oncogenicity study has been in progress since March 1982.

EPA (1984q) has stated that:

It was found that some studies on long-term effects performed by Industrial Bio-Test (IBT) Laboratories were invalid due to improper laboratory practices. In addition, a long-term study in rats sponsored by the National Cancer Institute is considered of questionable value due to laboratory procedures. The results of this rat study suggest that picloram may induce benign liver tumors. Even if this study were accepted as positive, given the high doses needed to produce the effect, and the very low potential for human exposure from current uses, existing uses would not pose a significant risk of increased cancer in the population. The registrant is conducting a new rat study to clarify the ambiguous results of this NCI study.

Simazine

In a preliminary oncogenicity study, the herbicide Fogard S^R (25 percent atrazine and 37.5 percent simazine) was administered to mice for a 7-month test period; as a result, malignant lymphomas were induced in test animals treated subcutaneously and intraperitoneally (Donna et al., 1981). As noted previously regarding atrazine, tumor formation in this study may have resulted from other chemicals in the formulation, or it may be related to the route of administration, because chronic simazine and atrazine feeding studies have shown no oncogenic effects, even at higher doses.

Appendix D
Human Health Risk
Assessment
(Quantitative)

Attachment B

Attachment B

DOSES TO WORKERS AND THE PUBLIC COMPUTED IN THE EXPOSURE ANALYSIS

Doses to workers for each of the application scenarios are given in mg/kg in the following tables. Doses to the public via each exposure route and for representative members of the public are given in micrograms/kg. Doses from accidental spraying to the public are given in micrograms/kg. Doses to workers and the public from spills are given in mg/kg.

Table B-1

Doses to Workers (mg/kg)
Routine-Realistic Aerial, 40 Acres by Helicopter

HERBICIDE	PILOT	MIXER/LOADER	SUPERVISOR	OBSERVER
AMITROLE	0.0004	0.0006	0.0001	0.0000
ASULAM	0.0483	0.0693	0.0074	0.0016
ATRAZINE	0.0755	0.1083	0.0116	0.0025
2,4-D	0.0302	0.0433	0.0046	0.0010
2,4-DP	0.0258	0.0370	0.0039	0.0008
DALAPON	0.0806	0.1156	0.0123	0.0026
DICAMBA	0.0101	0.0144	0.0015	0.0003
FOSAMINE	0.0604	0.0867	0.0092	0.0020
GLYPHOSATE	0.0403	0.0578	0.0062	0.0013
HEXAZINONE	0.0504	0.0722	0.0077	0.0016
PICLORAM	0.0010	0.0014	0.0001	0.0000
SIMAZINE	0.0806	0.1156	0.0123	0.0026
TEBUTHIURON	0.0201	0.0289	0.0031	0.0007
TRICLOPYR	0.0403	0.0578	0.0062	0.0013

Table B-2

Doses to Workers (mg/kg)
Large Aerial, 400 Acres by Fixed Wing, Routine-Worst Case

HERBICIDE	PILOT	MIXER/LOADER	SUPERVISOR	OBSERVER
AMITROLE	0.0067	0.0085	0.0012	0.0002
ASULAM	0.5591	0.7128	0.0969	0.0173
ATRAZINE	0.6696	0.8536	0.1160	0.0207
2,4-D	0.4018	0.5122	0.0696	0.0124
2,4-DP	0.2678	0.3414	0.0464	0.0083
DALAPON	1.6740	2.1340	0.2900	0.0517
DICAMBA	0.3348	0.4268	0.0580	0.0103
FOSAMINE	2.0088	2.5608	0.3480	0.0620
GLYPHOSATE	0.8370	1.0670	0.1450	0.0258
HEXAZINONE	0.5022	0.6402	0.0870	0.0155
PICLORAM	0.0402	0.0512	0.0070	0.0012
SIMAZINE	0.8370	1.0670	0.1450	0.0258
TEBUTHIURON	1.0044	1.2804	0.1740	0.0310
TRICLOPYR	1.3392	1.7072	0.2320	0.0413

Table B-3

Doses to Backpack Sprayers (mg/kg)
Small Backpack, 6.0 Acres, Routine-Realistic Case

HERBICIDE	DOSE
AMITROLE	0.0033
ASULAM	0.1978
ATRAZINE	0.4946
BROMACIL	0.6595
2,4-D	0.1978
2,4-DP	0.2110
DALAPON	0.6595
DICAMBA	0.0412
DIURON	0.6595
FOSAMINE	0.4946
GLYPHOSATE	0.2473
HEXAZINONE	0.1846
PICLORAM	0.0079
SIMAZINE	0.3297
TEBUTHIURON	0.2473
TRICLOPYR	0.3297

Table B-4

Doses to Backpack Sprayers (mg/kg)
Large Backpack, 60 Acres, Routine-Worst Case

HERBICIDE	DOSE
AMITROLE	0.0310
ASULAM	2.0693
ATRAZINE	2.4782
BROMACIL	6.1955
2,4-D	1.4869
2,4-DP	1.7050
DALAPON	7.4346
DICAMBA	1.2391
DIURON	3.7173
FOSAMINE	7.1248
GLYPHOSATE	3.0978
HEXAZINONE	1.8587
PICLORAM	0.1190
SIMAZINE	2.8499
TEBUTHIURON	3.7173
TRICLOPYR	4.9564

Table B-5

Doses to Workers (mg/kg)
Small Right of Way, Routine-Realistic Case

HERBICIDE	APPLICATOR	MIX/LOADER	APPL/MIX/LOADER
AMITROLE	0.0001	0.0001	0.0001
ASULAM	0.0082	0.0084	0.0116
ATRAZINE	0.0103	0.0105	0.0145
BROMACIL	0.0137	0.0140	0.0193
2,4-D	0.0051	0.0052	0.0072
2,4-DP	0.0055	0.0056	0.0077
DALAPON	0.0137	0.0140	0.0193
DICAMBA	0.0017	0.0017	0.0024
DIURON	0.0137	0.0140	0.0193
FOSAMINE	0.0137	0.0140	0.0193
GLYPHOSATE	0.0069	0.0070	0.0096
HEXAZINONE	0.0086	0.0087	0.0120
PICLORAM	0.0002	0.0002	0.0002
SIMAZINE	0.0069	0.0070	0.0096
TEBUTHIURON	0.0075	0.0077	0.0106
TRICLOPYR	0.0069	0.0070	0.0096

Table B-6

Doses to Workers (mg/kg)
Large Right of Way, Routine-Worst Case

HERBICIDE	APPLICATOR	MIX/LOADER	APPL/MIX/LOADER
AMITROLE	0.0042	0.0024	0.0029
ASULAM	0.2652	0.1530	0.1787
ATRAZINE	0.4509	0.2601	0.3038
BROMACIL	0.5305	0.3060	0.3574
2,4-D	0.1305	0.0753	0.0879
2,4-DP	0.1698	0.0979	0.1144
DALAPON	0.5305	0.3060	0.3574
DICAMBA	0.0955	0.0551	0.0643
DIURON	0.8488	0.4896	0.5719
FOSAMINE	0.5676	0.3274	0.3824
GLYPHOSATE	0.2652	0.1530	0.1787
HEXAZINONE	0.3183	0.1836	0.2145
PICLORAM	0.0051	0.0029	0.0034
SIMAZINE	0.2440	0.1408	0.1644
TEBUTHIURON	0.2440	0.1408	0.1644
TRICLOPYR	0.4244	0.2448	0.2859

Table B-7

Doses to Workers (mg/kg)
Hand Application to Small Site,
Routine Realistic

HERBICIDE	HACK & SQUIRT	INJECTION BAR
AMITROLE	0.00056	0.00021
BROMACIL	0.11134	0.04235
2,4-D	0.06680	0.02541
2,4-DP	0.10689	0.04066
DICAMBA	0.05567	0.02118
DIURON	0.11134	0.04235
FOSAMINE	0.11134	0.04235
PICLORAM	0.00267	0.00102
TRICLOPYR	0.11134	0.04235

Table B-8

Doses to Workers (mg/kg)
Hand Application to Large Site,
Routine-Worst Case

HERBICIDE	HACK & SQUIRT	INJECTION BAR
AMITROLE	0.00631	0.00169
BROMACIL	1.26170	0.33728
2,4-D	0.75702	0.20237
2,4-DP	1.21123	0.32379
DICAMBA	0.63085	0.16864
DIURON	1.26170	0.33728
FOSAMINE	1.26170	0.33728
PICLORAM	0.03028	0.00809
TRICLOPYR	1.26170	0.33728

Table B-9

Doses in micrograms/kg by Exposure Type:
Routine-Realistic Aerial, 40 Acres by Helicopter

HERBICIDE	SPRAY DRIFT DERMAL	VEGETA- TION CONTACT HIKER	VEGETA- TION CONTACT PICKER	DRINK WATER	EATING BERRIES	EATING VEGS.	EATING DEER	EATING BIRD	EATING FISH
AMITROLE	0.000	0.000	0.032	1.884	1.079	2.158	0.146	0.489	0.754
ASULAM	0.019	0.000	3.894	2.261	1.295	2.589	0.185	0.660	0.904
ATRAZINE	0.030	0.000	6.084	3.532	2.023	4.045	0.289	1.031	7.064
2,4-D	0.012	0.000	2.434	2.355	1.348	2.697	0.188	0.656	0.942
2,4-DP	0.010	0.000	2.077	1.884	1.079	2.158	0.151	0.528	0.754
DALAPON	0.032	0.000	6.490	3.768	2.158	4.315	0.308	1.100	1.507
DICAMBA	0.004	0.000	0.811	0.942	0.539	1.079	0.075	0.259	0.377
FOSAMINE	0.024	0.000	4.867	2.826	1.618	3.236	0.231	0.825	1.130
GLYPHOSATE	0.016	0.000	3.245	1.884	1.079	2.158	0.154	0.550	0.754
HEXAZINONE	0.020	0.000	4.056	2.355	1.348	2.697	0.192	0.687	0.942
PICLORAM	0.000	0.000	0.078	0.942	0.539	1.079	0.073	0.245	0.377
SIMAZINE	0.032	0.000	6.490	3.768	2.158	4.315	0.308	1.100	1.507
TEBUTHIURON	0.008	0.000	1.622	0.942	0.539	1.079	0.077	0.275	3.768
TRICLOPYR	0.016	0.000	3.245	1.884	1.079	2.158	0.154	0.550	0.754

Table B-10

Doses in micrograms/kg for Example People¹ for
Routine-Realistic Aerial, 40 Acres by Helicopter

HERBICIDE	HIKER	BERRY PICKER	HUNTER	FISHERMAN	NEARBY RESIDENT
AMITROLE	1.884	2.995	2.518	2.637	4.041
ASULAM	2.280	7.468	3.125	3.184	4.869
ATRAZINE	3.563	11.669	4.882	10.627	7.608
2,4-D	2.367	6.149	3.212	3.309	5.064
2,4-DP	1.894	5.049	2.573	2.648	4.052
DALAPON	3.800	12.447	5.208	5.307	8.115
DICAMBA	0.946	2.296	1.280	1.323	2.025
FOSAMINE	2.850	9.335	3.906	3.980	6.086
GLYPHOSATE	1.900	6.223	2.604	2.654	4.058
HEXAZINONE	2.375	7.779	3.255	3.317	5.072
PICLORAM	0.942	1.560	1.261	1.319	2.021
SIMAZINE	3.800	12.447	5.208	5.307	8.115
TEBUTHIURON	0.950	3.112	1.302	4.718	2.029
TRICLOPYR	1.900	6.223	2.604	2.654	4.058

¹All of these people receive multiple exposures as shown in Table 4-6.

Table B-11

Doses in micrograms/kg by Exposure Type for
Large Aerial, 400 Acres by Fixed Wing, Routine-Worst Case

HERBICIDE	SPRAY DRIFT DERMAL	VEGETA- TION CONTACT HIKER	VEGETA- TION CONTACT PICKER	DRINK WATER	EATING BERRIES	EATING VEGS.	EATING DEER	EATING BIRD	EATING FISH
AMITROLE	0.170	0.002	0.438	12.681	10.423	20.846	1.522	6.274	5.072
ASULAM	14.180	0.203	36.553	10.588	8.703	17.406	1.362	5.930	4.235
ATRAZINE	16.981	0.244	43.776	12.681	10.423	20.846	1.631	7.102	25.362
2,4-D	10.189	0.146	26.266	12.681	10.423	20.846	1.587	6.767	5.072
2,4-DP	6.793	0.097	17.510	7.926	6.514	13.028	0.994	4.251	3.170
DALAPON	42.454	0.609	109.440	31.702	26.057	52.114	4.077	17.754	12.681
DICAMBA	8.491	0.122	21.888	12.681	10.423	20.846	1.576	6.684	5.072
FOSAMINE	50.944	0.731	131.328	38.042	31.268	62.537	4.892	21.305	15.217
GLYPHOSATE	21.227	0.305	54.720	15.851	13.028	26.057	2.039	8.877	6.340
HEXAZINONE	12.736	0.183	32.832	9.511	7.817	15.634	1.223	5.326	3.804
PICLORAM	1.019	0.015	2.627	15.851	13.028	26.057	1.907	7.883	6.340
SIMAZINE	21.227	0.305	54.720	15.851	13.028	26.057	2.039	8.877	6.340
TEBUTHIURON	25.472	0.365	65.664	19.021	15.634	31.268	2.446	10.653	76.085
TRICLOPYR	33.963	0.487	87.552	25.362	20.846	41.691	3.262	14.203	10.145

Table B-12

Doses in micrograms/kg for Example People¹ for
Large Aerial, 400 Acres by Fixed Wing, Routine-Worst Case

HERBICIDE	HIKER	BERRY PICKER	HUNTER	FISHERMAN	NEARBY RESIDENT
AMITROLE	12.853	23.711	20.649	17.925	33.699
ASULAM	24.971	70.024	32.263	29.207	42.377
ATRAZINE	29.906	83.861	38.638	55.267	50.751
2,4-D	23.016	59.558	31.370	28.088	43.861
2,4-DP	14.816	38.743	20.061	17.986	27.844
DALAPON	74.765	209.653	96.596	87.445	126.879
DICAMBA	21.293	53.482	29.553	26.366	42.139
FOSAMINE	89.718	251.583	115.915	104.935	152.254
GLYPHOSATE	37.382	104.826	48.298	43.723	63.439
HEXAZINONE	22.429	62.896	28.979	26.234	38.064
PICLORAM	16.885	32.525	26.674	23.225	42.941
SIMAZINE	37.382	104.826	48.298	43.723	63.439
TEBUTHIURON	44.859	125.792	57.958	120.944	76.127
TRICLOPYR	59.812	167.722	77.277	69.956	101.503

¹All of these people receive multiple exposures as shown in Table 4-6.

Table B-13

Doses in micrograms/kg by Exposure Type for
Small Backpack, 6.0 Acres, Routine-Realistic Case

HERBICIDE	SPRAY DRIFT DERMAL	VEGETA- TION CONTACT HIKER	VEGETA- TION CONTACT PICKER	DRINK WATER	EATING BERRIES	EATING VEGS.	EATING DEER	EATING BIRD	EATING FISH
AMITROLE	0.002	0.000	0.007	0.166	0.306	0.612	0.038	0.109	0.066
ASULAM	0.107	0.002	0.423	0.100	0.184	0.367	0.024	0.073	0.040
ATRAZINE	0.267	0.004	1.057	0.249	0.459	0.918	0.060	0.184	0.498
BROMACIL	0.356	0.005	1.409	0.332	0.612	1.224	0.080	0.245	0.133
2,4-D	0.107	0.002	0.423	0.166	0.306	0.612	0.039	0.117	0.066
2,4-DP	0.114	0.002	0.451	0.166	0.306	0.612	0.039	0.118	0.066
DALAPON	0.356	0.005	1.409	0.332	0.612	1.224	0.080	0.245	0.133
DICAMBA	0.022	0.000	0.088	0.041	0.076	0.153	0.010	0.029	0.017
DIURON	0.356	0.005	1.409	0.332	0.612	1.224	0.080	0.245	2.654
FOSAMINE	0.267	0.004	1.057	0.249	0.459	0.918	0.060	0.184	0.100
GLYPHOSATE	0.134	0.002	0.528	0.124	0.229	0.459	0.030	0.092	0.050
HEXAZINONE	0.100	0.001	0.395	0.093	0.171	0.343	0.022	0.069	0.037
PICLORAM	0.004	0.000	0.017	0.083	0.153	0.306	0.019	0.055	0.033
SIMAZINE	0.178	0.003	0.705	0.166	0.306	0.612	0.040	0.122	0.066
TEBUTHIURON	0.134	0.002	0.528	0.124	0.229	0.459	0.030	0.092	0.498
TRICLOPYR	0.178	0.003	0.705	0.166	0.306	0.612	0.040	0.122	0.066

Table B-14

Doses in micrograms/kg for Example People¹ for
Small Backpack, 6.0 Acres, Routine-Realistic Case

HERBICIDE	HIKER	BERRY PICKER	HUNTER	FISHERMAN	NEARBY RESIDENT
AMITROLE	0.168	0.481	0.315	0.234	0.780
ASULAM	0.208	0.813	0.305	0.248	0.575
ATRAZINE	0.520	2.032	0.763	1.017	1.438
BROMACIL	0.693	2.709	1.017	0.825	1.917
2,4-D	0.274	1.001	0.430	0.341	0.886
2,4-DP	0.281	1.037	0.438	0.348	0.893
DALAPON	0.693	2.709	1.017	0.825	1.917
DICAMBA	0.064	0.228	0.103	0.081	0.217
DIURON	0.693	2.709	1.017	3.346	1.917
FOSAMINE	0.520	2.032	0.763	0.619	1.438
GLYPHOSATE	0.260	1.016	0.381	0.310	0.719
HEXAZINONE	0.194	0.759	0.285	0.231	0.537
PICLORAM	0.087	0.257	0.161	0.120	0.393
SIMAZINE	0.346	1.354	0.509	0.413	0.958
TEBUTHIURON	0.260	1.016	0.381	0.757	0.719
TRICLOPYR	0.346	1.354	0.509	0.413	0.958

¹All of these people receive multiple exposures as shown in Table 4-6.

Table B-15

Doses in micrograms/kg by Exposure Type for
Large Backpack, 60 Acres, Routine-Worst Case

HERBICIDE	SPRAY DRIFT DERMAL	VEGETA- TION CONTACT HIKER	VEGETA- TION CONTACT PICKER	DRINK WATER	EATING BERRIES	EATING VEGS.	EATING DEER	EATING BIRD	EATING FISH
AMITROLE	0.009	0.000	0.022	0.477	0.922	1.843	0.116	0.339	0.191
ASULAM	0.572	0.008	1.475	0.319	0.616	1.231	0.081	0.255	0.128
ATRAZINE	0.685	0.010	1.766	0.382	0.737	1.475	0.097	0.305	0.764
BROMACIL	1.713	0.025	4.416	0.955	1.843	3.687	0.242	0.762	0.382
2,4-D	0.411	0.006	1.060	0.382	0.737	1.475	0.095	0.291	0.153
2,4-DP	0.471	0.007	1.215	0.411	0.793	1.585	0.102	0.315	0.164
DALAPON	2.056	0.029	5.299	1.146	2.212	4.424	0.290	0.915	0.458
DICAMBA	0.343	0.005	0.883	0.382	0.737	1.475	0.095	0.288	0.153
DIURON	1.028	0.015	2.650	0.573	1.106	2.212	0.145	0.457	4.584
FOSAMINE	1.970	0.028	5.078	1.098	2.120	4.240	0.278	0.876	0.439
GLYPHOSATE	0.857	0.012	2.208	0.477	0.922	1.843	0.121	0.381	0.191
HEXAZINONE	0.514	0.007	1.325	0.286	0.553	1.106	0.073	0.229	0.115
PICLORAM	0.033	0.000	0.085	0.382	0.737	1.475	0.093	0.273	0.153
SIMAZINE	0.788	0.011	2.031	0.439	0.848	1.696	0.111	0.351	0.176
TEBUTHIURON	1.028	0.015	2.650	0.573	1.106	2.212	0.145	0.457	2.292
TRICLOPYR	1.370	0.020	3.533	0.764	1.475	2.949	0.194	0.610	0.306

Table B-16

Doses in micrograms/kg for Example People¹ for
Large Backpack, 60 Acres, Routine-Worst Case

HERBICIDE	HIKER	BERRY PICKER	HUNTER	FISHERMAN	NEARBY RESIDENT
AMITROLE	0.486	1.430	0.941	0.677	2.330
ASULAM	0.899	2.982	1.235	1.027	2.131
ATRAZINE	1.077	3.571	1.479	1.841	2.552
BROMACIL	2.693	8.927	3.697	3.075	6.379
2,4-D	0.799	2.590	1.185	0.952	2.274
2,4-DP	0.889	2.890	1.306	1.053	2.474
DALAPON	3.231	10.713	4.436	3.690	7.655
DICAMBA	0.730	2.345	1.112	0.882	2.204
DIURON	1.616	5.356	2.218	6.200	3.828
FOSAMINE	3.096	10.267	4.251	3.536	7.336
GLYPHOSATE	1.346	4.464	1.848	1.537	3.190
HEXAZINONE	0.808	2.678	1.109	0.922	1.914
PICLORAM	0.415	1.237	0.781	0.568	1.890
SIMAZINE	1.239	4.107	1.701	1.414	2.935
TEBUTHIURON	1.616	5.356	2.218	3.908	3.228
TRICLOPYR	2.154	7.142	2.957	2.460	5.103

¹All of these people receive multiple exposures as shown in Table 4-6.

Table B-17

Doses in micrograms/kg by Exposure Type for
Small Right of Way, Routine-Realistic Case

HERBICIDE	SPRAY DRIFT DERMAL	VEGETA- TION CONTACT HIKER	VEGETA- TION CONTACT PICKER	DRINK WATER	EATING BERRIES	EATING VEGS.	EATING DEER	EATING BIRD	EATING FISH
AMITROLE	0.000	0.000	0.002	0.074	0.102	0.204	0.012	0.030	0.030
ASULAM	0.041	0.001	0.224	0.089	0.123	0.245	0.015	0.041	0.036
ATRAZINE	0.052	0.001	0.280	0.112	0.153	0.307	0.019	0.051	0.223
BROMACIL	0.069	0.001	0.373	0.149	0.204	0.409	0.025	0.068	0.060
2,4-D	0.026	0.000	0.140	0.093	0.128	0.256	0.016	0.041	0.037
2,4-DP	0.028	0.000	0.149	0.093	0.128	0.256	0.016	0.041	0.037
DALAPON	0.069	0.001	0.373	0.149	0.204	0.409	0.025	0.068	0.060
DICAMBA	0.009	0.000	0.047	0.037	0.051	0.102	0.006	0.016	0.015
DIURON	0.069	0.001	0.373	0.149	0.204	0.409	0.025	0.068	1.191
FOSAMINE	0.069	0.001	0.373	0.149	0.204	0.409	0.025	0.068	0.060
GLYPHOSATE	0.034	0.000	0.187	0.074	0.102	0.204	0.013	0.034	0.030
HEXAZINONE	0.043	0.001	0.233	0.093	0.128	0.256	0.016	0.042	0.037
PICLORAM	0.001	0.000	0.004	0.037	0.051	0.102	0.006	0.015	0.015
SIMAZINE	0.034	0.000	0.187	0.074	0.102	0.204	0.013	0.034	0.030
TEBUTHIURON	0.038	0.001	0.205	0.082	0.112	0.225	0.014	0.037	0.327
TRICLOPYR	0.034	0.000	0.187	0.074	0.102	0.204	0.013	0.034	0.030

Table B-18

Doses in micrograms/kg for Example People¹ for
Small Right of Way, Routine-Realistic Case

HERBICIDE	HIKER	BERRY PICKER	HUNTER	FISHERMAN	NEARBY RESIDENT
AMITROLE	0.075	0.179	0.117	0.105	0.279
ASULAM	0.131	0.477	0.187	0.167	0.377
ATRAZINE	0.164	0.597	0.234	0.387	0.471
BROMACIL	0.219	0.796	0.312	0.278	0.628
2,4-D	0.119	0.387	0.175	0.156	0.375
2,4-DP	0.121	0.398	0.177	0.158	0.377
DALAPON	0.219	0.796	0.312	0.278	0.628
DICAMBA	0.046	0.144	0.068	0.061	0.148
DIURON	0.219	0.796	0.312	1.409	0.628
FOSAMINE	0.219	0.796	0.312	0.278	0.628
GLYPHOSATE	0.109	0.398	0.156	0.139	0.314
HEXAZINONE	0.137	0.497	0.195	0.174	0.392
PICLORAM	0.038	0.094	0.059	0.053	0.140
SIMAZINE	0.109	0.398	0.156	0.139	0.314
TEBUTHIURON	0.120	0.438	0.172	0.448	0.345
TRICLOPYR	0.109	0.398	0.156	0.139	0.314

¹All of these people receive multiple exposures as shown in Table 4-6.

Table B-19

Doses in micrograms/kg by Exposure Type for
Large Right of Way, Routine-Worst Case

HERBICIDE	SPRAY DRIFT DERMAL	VEGETA- TION CONTACT HIKER	VEGETA- TION CONTACT PICKER	DRINK WATER	EATING BERRIES	EATING VEGS.	EATING DEER	EATING BIRD	EATING FISH
AMITROLE	0.005	0.000	0.012	0.398	0.620	1.240	0.075	0.197	0.159
ASULAM	0.300	0.004	0.773	0.249	0.388	0.775	0.049	0.138	0.100
ATRAZINE	0.510	0.007	1.314	0.423	0.659	1.318	0.083	0.234	0.846
BROMACIL	0.600	0.009	1.546	0.498	0.775	1.550	0.098	0.275	0.199
2,4-D	0.147	0.002	0.380	0.204	0.318	0.636	0.040	0.108	0.082
2,4-DP	0.192	0.003	0.495	0.249	0.388	0.775	0.048	0.132	0.100
DALAPON	0.600	0.009	1.546	0.498	0.775	1.550	0.098	0.275	0.199
DICAMBA	0.108	0.002	0.278	0.179	0.279	0.558	0.035	0.094	0.072
DIURON	0.959	0.014	2.473	0.796	1.240	2.481	0.157	0.441	6.372
FOSAMINE	0.642	0.009	1.654	0.533	0.829	1.659	0.105	0.295	0.213
GLYPHOSATE	0.300	0.004	0.773	0.249	0.388	0.775	0.049	0.138	0.100
HEXAZINONE	0.360	0.005	0.927	0.299	0.465	0.930	0.059	0.165	0.119
PICLORAM	0.006	0.000	0.015	0.100	0.155	0.310	0.019	0.049	0.040
SIMAZINE	0.276	0.004	0.711	0.229	0.357	0.713	0.045	0.127	0.092
TEBUTHIURON	0.276	0.004	0.711	0.229	0.357	0.713	0.045	0.127	0.916
TRICLOPYR	0.480	0.007	1.236	0.398	0.620	1.240	0.078	0.220	0.159

Table B-20

Doses in micrograms/kg for Example People¹ for
Large Right of Way, Routine-Worst Case

HERBICIDE	HIKER	BERRY PICKER	HUNTER	FISHERMAN	NEARBY RESIDENT
AMITROLE	0.403	1.036	0.675	0.562	1.643
ASULAM	0.553	1.709	0.740	0.653	1.328
ATRAZINE	0.940	2.905	1.258	1.786	2.258
BROMACIL	1.106	3.418	1.479	1.305	2.656
2,4-D	0.354	1.050	0.501	0.435	0.989
2,4-DP	0.444	1.323	0.624	0.543	1.219
DALAPON	1.106	3.418	1.479	1.305	2.656
DICAMBA	0.289	0.844	0.417	0.360	0.847
DIURON	1.770	5.469	2.367	8.141	4.250
FOSAMINE	1.183	3.657	1.583	1.396	2.842
GLYPHOSATE	0.553	1.709	0.740	0.653	1.328
HEXAZINONE	0.664	2.051	0.888	0.783	1.594
PICLORAM	0.105	0.275	0.174	0.145	0.415
SIMAZINE	0.509	1.572	0.681	0.600	1.222
TEBUTHIURON	0.509	1.572	0.681	1.425	1.222
TRICLOPYR	0.885	2.735	1.184	1.044	2.125

¹All of these people receive multiple exposures as shown in Table 4-6.

Table B-21

Doses in micrograms/kg from Items Receiving the Full Per Acre
Application Rate by Exposure Type
Accidental-Worst Case

HERBICIDE	DIRECT DERMAL	REENTRY HIKER	REENTRY PICKER	DRINK WATER	EATING BERRIES	EATING VEGS.	EATING DEER	EATING BIRD	EATING FISH
AMITROLE	3.	0.	9.	117.	93.	194.	19.	116.	47.
ASULAM	209.	3.	538.	73.	58.	121.	13.	83.	29.
ATRAZINE	355.	5.	914.	125.	99.	206.	22.	141.	249.
BROMACIL	417.	6.	1075.	147.	116.	242.	26.	165.	59.
2,4-D	103.	1.	264.	60.	48.	99.	10.	64.	24.
2,4-DP	133.	2.	344.	73.	58.	121.	13.	79.	29.
DALAPON	417.	6.	1075.	147.	116.	242.	26.	165.	59.
DICAMBA	83.	1.	215.	59.	47.	97.	10.	62.	23.
DIURON	667.	10.	1720.	235.	186.	387.	42.	265.	1878.
FOSAMINE	501.	7.	1290.	176.	140.	290.	31.	199.	70.
GLYPHOSATE	209.	3.	538.	73.	58.	121.	13.	83.	29.
HEXAZINONE	250.	4.	645.	88.	70.	145.	16.	99.	35.
PICLORAM	10.	0.	26.	73.	58.	121.	12.	73.	29.
SIMAZINE	209.	3.	538.	73.	58.	121.	13.	83.	29.
TEBUTHIURON	250.	4.	645.	88.	70.	145.	16.	99.	352.
TRICLOPYR	334.	5.	860.	117.	93.	194.	21.	132.	47.

Table B-22

Doses in micrograms/kg for Example People¹ for
Accidental-Worst Case Spraying

HERBICIDE	HIKER	BERRY PICKER	HUNTER	FISHERMAN	NEARBY RESIDENT
AMITROLE	121.	222.	256.	168.	314.
ASULAM	285.	878.	381.	314.	406.
ATRAZINE	484.	1492.	647.	734.	690.
BROMACIL	570.	1755.	761.	628.	812.
2,4-D	164.	475.	239.	188.	263.
2,4-DP	209.	609.	300.	238.	330.
DALAPON	570.	1755.	761.	628.	812.
DICAMBA	143.	404.	215.	167.	240.
DIURON	912.	2809.	1218.	2790.	1299.
FOSAMINE	684.	2107.	914.	754.	974.
GLYPHOSATE	285.	878.	381.	314.	406.
HEXAZINONE	342.	1053.	457.	377.	487.
PICLORAM	84.	167.	168.	113.	204.
SIMAZINE	285.	878.	381.	314.	406.
TEBUTHIURON	342.	1053.	457.	694.	487.
TRICLOPYR	456.	1404.	609.	503.	649.

¹All of these people receive multiple exposures as shown in Table 4-6.

Table B-23

Doses from Herbicide Spills (mg/kg)

HERBICIDE	SPILL OF ONE PINT CONCENTRATE ON SKIN	SPILL OF ONE PINT TANK MIX ON SKIN	HELICOPTER ¹ DUMP INTO POND	HELICOPTER ¹ DUMP INTO RESERVOIR	TRUCK ¹ SPILL INTO POND	TRUCK ¹ SPILL INTO RESERVOIR
AMITROLE	1.20	0.24	0.0737	0.0023	1.4730	0.0460
ASULAM	240.00	20.04	0.0615	0.0019	1.2300	0.0384
ATRAZINE	240.00	24.00	0.0737	0.0023	1.4730	0.0460
BROMACIL	240.00	12.00	-----	-----	0.7365	0.0230
2,4-D	144.00	14.40	0.0737	0.0023	1.4730	0.0460
2,4-DP	230.40	9.60	0.0460	0.0014	0.9206	0.0288
DALAPON	---	60.00	0.1841	0.0058	3.6825	0.1151
DICAMBA	120.00	12.00	0.0737	0.0023	1.4730	0.0460
DIURON	240.00	19.20	-----	-----	1.1784	0.0368
FOSAMINE	240.00	72.00	0.2210	0.0069	4.4191	0.1381
GLYPHOSATE	180.00	30.00	0.0921	0.0029	1.8413	0.0575
HEXAZINONE	120.00	18.00	0.0552	0.0017	1.1048	0.0345
PICLORAM	5.76	1.44	0.0921	0.0029	1.8413	0.0575
SIMAZINE	240.00	30.00	0.0921	0.0029	1.8413	0.0575
TEBUTHIURON	---	36.00	0.1105	0.0035	2.2095	0.0690
TRICLOPYR	240.00	48.00	0.1473	0.0046	2.9460	0.0921

¹Assuming 1 liter of water drunk per day.

Table B-24

Lifetime Doses for Exposed Workers (mg/kg/day)

Exposures per Lifetime	Average Daily Dose for Exclusive Use of						Glyphosate	
	2,4-D	2,4-DP	Asulam	Bromacil	Picloram	Amitrole		
Realistic Number of Exposures								
Pilot	30	5.73E-05	4.45E-05	8.68E-05	----	3.44E-06	8.43E-07	9.41E-05
Mixer/Loader	30	7.84E-05	6.13E-05	1.19E-04	----	4.55E-06	1.15E-06	1.27E-04
Supervisor	30	9.24E-06	7.12E-06	1.39E-05	----	5.74E-07	1.37E-07	1.54E-05
Observer	30	1.82E-06	1.42E-06	2.76E-06	----	1.08E-07	2.67E-08	2.97E-06
Backpack	50	5.13E-04	5.59E-04	5.70E-04	1.83E-03	2.64E-05	9.16E-06	7.63E-04
R-O-W Sprayer	45	2.01E-05	2.41E-05	3.71E-05	6.97E-05	7.24E-07	4.88E-07	3.48E-05
R-O-W Mix/L	45	1.54E-05	1.80E-05	2.75E-05	5.03E-05	5.39E-07	3.32E-07	2.52E-05
R-O-W AP/M/L	45	1.98E-05	2.30E-05	3.51E-05	6.37E-05	6.89E-07	4.13E-07	3.19E-05
Hack & Squirt	70	2.78E-04	4.44E-04	----	4.63E-04	1.11E-05	2.31E-06	----
Injection Bar	70	9.39E-05	1.50E-04	----	1.56E-04	3.75E-06	7.82E-07	----
Worst Case Number of Exposures								
Pilot	288	5.50E-04	4.27E-04	8.33E-04	----	3.30E-05	8.09E-06	9.03E-04
Mixer/Loader	288	7.53E-04	5.88E-04	1.14E-03	----	4.37E-05	1.10E-05	1.22E-03
Supervisor	288	8.87E-05	6.84E-05	1.34E-04	----	5.51E-06	1.31E-06	1.48E-04
Observer	288	1.75E-05	1.36E-05	2.65E-05	----	1.03E-06	2.56E-07	2.86E-05
Backpack	440	4.52E-03	4.92E-03	5.02E-03	1.61E-02	2.32E-04	8.06E-05	6.71E-03
R-O-W Sprayer	416	1.86E-04	2.23E-04	3.43E-04	6.44E-04	6.69E-06	4.52E-06	3.22E-04
R-O-W Mix/L	416	1.42E-04	1.66E-04	2.54E-04	4.65E-04	4.98E-06	3.07E-06	2.33E-04
R-O-W AP/M/L	416	1.83E-04	2.12E-04	3.24E-04	5.89E-04	6.37E-06	3.82E-06	2.95E-04
Hack & Squirt	480	1.90E-03	3.05E-03	----	3.17E-03	7.61E-05	1.59E-05	----
Injection Bar	480	6.44E-04	1.03E-03	----	1.07E-03	2.57E-05	5.36E-06	----

Table B-26
Lifetime Doses for Exposed Public (mg/kg/day)
Large Aerial, 400 Acres by Fixed Wing, Worst Case

Exposures per Lifetime	Average Daily Dose for Exclusive Use of					
	2,4-D	2,4-DP	Asulam	Bromacil	Picloram	Amitrole
<u>For a Single Exposure</u>						
Dermal, Spray	3.99E-07	2.66E-07	5.55E-07	---	3.99E-08	6.65E-09
Vegetation Contact						
Hiker	1					8.31E-07
Picker	1	3.81E-09	7.96E-09	---	5.72E-10	9.54E-11
Drinking Water	1	6.85E-07	1.43E-06	---	1.03E-07	1.71E-08
Eating Berries	1	3.10E-07	4.14E-07	---	6.20E-07	4.96E-07
Eating Vegets.	1	2.55E-07	3.41E-07	---	5.10E-07	4.08E-07
Eating Deer	1	5.10E-07	6.81E-07	---	1.02E-06	8.16E-07
Eating Fish	1	3.89E-08	5.33E-08	---	7.46E-08	5.96E-08
	1	1.24E-07	1.66E-07	---	2.48E-07	1.99E-07
<u>Combined Routes of Exposure</u>						
Hiker	1	5.80E-07	9.77E-07	---	6.61E-07	5.03E-07
Berry Picker	1	2.33E-06	2.74E-06	---	1.27E-06	9.28E-07
Hunter	1	1.23E-06	1.26E-06	---	1.04E-06	8.08E-07
Fisherman	1	1.10E-06	1.14E-06	---	9.09E-07	7.02E-07
Resident	1	1.72E-06	1.66E-06	---	1.68E-06	1.32E-06
<u>For 30 Exposures</u>						
Dermal, Spray	1.20E-05	7.98E-06	1.66E-05	---	1.20E-06	1.99E-07
Vegetation Contact						
Hiker	30					2.49E-05
Picker	30	1.72E-07	2.39E-07	---	1.72E-08	2.86E-09
Drinking Water	30	3.08E-05	4.29E-05	---	3.08E-06	5.14E-07
Eating Berries	30	1.49E-05	1.24E-05	---	1.86E-05	1.49E-05
Eating Vegets.	30	1.22E-05	1.02E-05	---	1.53E-05	1.22E-05
Eating Deer	30	2.45E-05	2.04E-05	---	3.06E-05	2.45E-05
Eating Fish	30	1.86E-06	1.60E-06	---	2.24E-06	1.79E-06
	30	5.96E-06	4.97E-06	---	7.44E-06	5.96E-06
<u>Combined Routes of Exposure</u>						
Hiker	30	2.70E-05	2.93E-05	---	1.98E-05	1.51E-05
Berry Picker	30	6.99E-05	8.22E-05	---	3.82E-05	2.78E-05
Hunter	30	3.68E-05	3.79E-05	---	3.13E-05	2.42E-05
Fisherman	30	3.30E-05	3.43E-05	---	2.73E-05	2.10E-05
Resident	30	5.15E-05	4.98E-05	---	5.04E-05	3.96E-05
						4.39E-05
						1.23E-04
						5.67E-05
						5.13E-05
						7.45E-05

Table B-27
Lifetime Doses for Exposed Public (mg/kg/day)
Small Backpack, 6.0 Acres, Reaslitic Case

Exposures per Lifetime	Average Daily Dose for Exclusive Use of					
	2,4-D	2,4-DP	Asulam	Bromacil	Picloram	Amitrole
<u>For a Single Exposure</u>						
Dermal, Spray	4.18E-09	4.46E-09	4.18E-09	1.39E-08	1.67E-10	6.97E-11
Vegetation Contact						
Hiker	6.00E-11	6.40E-11	6.00E-11	2.00E-10	2.40E-12	1.00E-12
Picker	1.65E-08	1.77E-08	1.65E-08	5.52E-08	6.62E-10	2.76E-10
Drinking Water	6.49E-09	6.49E-09	3.89E-09	1.30E-08	3.25E-09	6.49E-09
Eating Berries	1.20E-08	1.20E-08	7.19E-09	2.40E-08	5.99E-09	1.20E-08
Eating Vegets.	2.40E-08	2.40E-08	1.44E-08	4.79E-08	1.20E-08	2.40E-08
Eating Deer	1.53E-09	1.53E-09	9.35E-10	3.12E-09	7.46E-10	1.49E-09
Eating Fish	2.60E-09	2.60E-09	1.56E-09	5.19E-09	1.30E-09	2.60E-09
<u>Combined Routes of Exposure</u>						
Hiker	1.07E-08	1.10E-08	8.13E-09	2.71E-08	3.42E-09	6.56E-09
Berry Picker	3.92E-08	4.06E-08	3.18E-08	1.06E-07	1.01E-08	1.88E-08
Hunter	1.68E-08	1.71E-08	1.19E-08	3.98E-08	6.31E-09	1.23E-08
Fisherman	1.33E-08	1.36E-08	9.69E-09	3.23E-08	4.71E-09	9.16E-09
Resident	3.47E-08	3.50E-08	2.25E-08	7.50E-08	1.54E-08	3.05E-08
<u>For 30 Exposures</u>						
Dermal, Spray	1.25E-07	1.34E-07	1.25E-07	4.18E-07	5.02E-09	2.09E-09
Vegetation Contact						
Hiker	1.80E-09	1.92E-09	1.80E-09	6.00E-09	7.20E-11	3.00E-11
Picker	4.96E-07	5.30E-07	4.96E-07	1.65E-06	1.99E-08	8.27E-09
Drinking Water	1.95E-07	1.95E-07	1.17E-07	3.89E-07	9.74E-08	1.95E-07
Eating Berries	3.59E-07	3.59E-07	2.16E-07	7.19E-07	1.80E-07	3.59E-07
Eating Vegets.	7.19E-07	7.19E-07	4.31E-07	1.44E-06	3.59E-07	7.19E-07
Eating Deer	4.59E-08	4.60E-08	2.81E-08	9.35E-08	2.24E-08	4.47E-08
Eating Fish	7.79E-08	7.79E-08	4.67E-08	1.56E-07	3.89E-08	7.79E-08
<u>Combined Routes of Exposure</u>						
Hiker	3.22E-07	3.30E-07	2.44E-07	8.13E-07	1.02E-07	1.97E-07
Berry Picker	1.18E-06	1.22E-06	9.54E-07	3.18E-06	3.02E-07	5.64E-07
Hunter	5.05E-07	5.14E-07	3.58E-07	1.19E-06	1.89E-07	3.70E-07
Fisherman	4.00E-07	4.08E-07	2.91E-07	9.69E-07	1.41E-07	2.75E-07
Resident	1.04E-06	1.05E-06	6.75E-07	2.25E-06	4.62E-07	9.15E-07
						8.44E-07
						3.05E-07
						1.19E-06
						4.48E-07
						3.63E-07
						8.44E-07
						5.84E-08
						3.51E-08
						5.39E-07
						2.69E-07
						1.46E-07
						6.21E-07
						2.25E-09
						1.57E-07

Table B-28
Lifetime Doses for Exposed Public (mg/kg/day)
Large Backpack, 60 Acres, Worst Case

Exposures per Lifetime	Average Daily Dose for Exclusive Use of						Glyphosate	
	2,4-D	2,4-DP	Asulam	Bromacil	Picloram	Amitrole		
For a Single Exposure								
Dermal, Spray	1	1.61E-08	1.85E-08	2.24E-08	6.70E-08	1.29E-09	3.35E-10	3.35E-08
Vegetation Contact	1	2.31E-10	2.65E-10	3.21E-10	9.62E-10	1.85E-11	4.81E-12	4.81E-10
Hiker	1	4.15E-08	4.76E-08	5.77E-08	1.73E-07	3.32E-09	8.64E-10	8.64E-08
Picker	1	1.50E-08	1.61E-08	1.25E-08	3.74E-08	1.50E-08	1.87E-08	1.87E-08
Drinking Water	1	2.89E-08	3.10E-08	2.41E-08	7.21E-08	2.89E-08	3.61E-08	3.61E-08
Eating Berries	1	5.77E-08	6.20E-08	4.82E-08	1.44E-07	5.77E-08	7.21E-08	7.21E-08
Eating Vegets.	1	3.72E-09	4.01E-09	3.16E-09	9.47E-09	3.62E-09	4.52E-09	4.74E-09
Eating Deer	1	5.98E-09	6.43E-09	4.99E-09	1.50E-08	5.98E-09	7.48E-09	7.48E-09
Eating Fish	1							
Combined Routes of Exposure								
Hiker	1	3.13E-08	3.48E-08	3.52E-08	1.05E-07	1.63E-08	1.90E-08	5.27E-08
Berry Picker	1	1.01E-07	1.13E-07	1.17E-07	3.49E-07	4.84E-08	5.60E-08	1.75E-07
Hunter	1	4.64E-08	5.11E-08	4.83E-08	1.45E-07	3.06E-08	3.68E-08	7.23E-08
Fisherman	1	3.73E-08	4.12E-08	4.02E-08	1.20E-07	2.22E-08	2.65E-08	6.02E-08
Resident	1	8.90E-08	9.68E-08	8.34E-08	2.50E-07	7.40E-08	9.12E-08	1.25E-07
For 30 Exposures								
Dermal, Spray	30	4.83E-07	5.54E-07	6.72E-07	2.01E-06	3.86E-08	1.01E-08	1.01E-06
Vegetation Contact	30	6.93E-09	7.94E-09	9.64E-09	2.89E-08	5.54E-10	1.44E-10	1.44E-08
Hiker	30	1.24E-06	1.43E-06	1.73E-06	5.19E-06	9.96E-08	2.59E-08	2.59E-06
Picker	30	4.49E-07	4.82E-07	3.75E-07	1.12E-06	4.49E-07	5.61E-07	5.61E-07
Drinking Water	30	8.66E-07	9.31E-07	7.23E-07	2.16E-06	8.66E-07	1.08E-06	1.08E-06
Eating Berries	30	1.73E-06	1.86E-06	1.45E-06	4.33E-06	1.73E-06	2.16E-06	2.16E-06
Eating Vegets.	30	1.12E-07	1.20E-07	9.49E-08	2.84E-07	1.09E-07	1.36E-07	1.42E-07
Eating Deer	30	1.79E-07	1.93E-07	1.50E-07	4.49E-07	1.79E-07	2.24E-07	2.24E-07
Eating Fish	30							
Combined Routes of Exposure								
Hiker	30	9.38E-07	1.04E-06	1.06E-06	3.16E-06	4.88E-07	5.71E-07	1.58E-06
Berry Picker	30	3.04E-06	3.39E-06	3.50E-06	1.05E-05	1.45E-06	1.68E-06	5.24E-06
Hunter	30	1.39E-06	1.53E-06	1.45E-06	4.34E-06	9.17E-07	1.10E-06	2.17E-06
Fisherman	30	1.12E-06	1.24E-06	1.21E-06	3.61E-06	6.67E-07	7.95E-07	1.81E-06
Resident	30	2.67E-06	2.91E-06	2.50E-06	7.49E-06	2.22E-06	2.74E-06	3.75E-06

Table B-29
Lifetime Doses for Exposed Public (mg/kg/day)
Small Right of Way, Realistic Case

Exposures per Lifetime	Average Daily Dose for Exclusive Use of					
	2,4-D	2,4-DP	Asulam	Bromacil	Picloram	Amitrole
Glyphosate						
<u>For a Single Exposure</u>						
Dermal, Spray	1.01E-09	1.08E-09	1.62E-09	2.70E-09	3.24E-11	1.35E-11
Vegetation Contact						
Hiker	1.45E-11	1.55E-11	2.32E-11	3.87E-11	4.65E-13	1.94E-13
Picker	5.48E-09	5.84E-09	8.77E-09	1.46E-08	1.75E-10	7.30E-11
Drinking Water	3.64E-09	3.64E-09	3.49E-09	5.82E-09	1.46E-09	2.91E-09
Eating Berries	5.00E-09	5.00E-09	4.80E-09	8.00E-09	2.00E-09	4.00E-09
Eating Vegets.	1.00E-08	1.00E-08	9.60E-09	1.60E-08	4.00E-09	8.00E-09
Eating Deer	6.14E-10	6.15E-10	5.98E-10	9.97E-10	2.41E-10	4.80E-10
Eating Fish	1.46E-09	1.46E-09	1.40E-09	2.33E-09	5.82E-10	1.16E-09
<u>Combined Routes of Exposure</u>						
Hiker	4.67E-09	4.74E-09	5.14E-09	8.56E-09	1.49E-09	2.93E-09
Berry Picker	1.51E-08	1.56E-08	1.87E-08	3.11E-08	3.66E-09	7.00E-09
Hunter	6.87E-09	6.94E-09	7.33E-09	1.22E-08	2.33E-09	4.59E-09
Fisherman	6.12E-09	6.19E-09	6.54E-09	1.09E-08	2.07E-09	4.09E-09
Resident	1.47E-08	1.47E-08	1.47E-08	2.46E-08	5.49E-09	1.09E-08
<u>For 30 Exposures</u>						
Dermal, Spray	3.04E-08	3.24E-08	4.86E-08	8.10E-08	9.72E-10	4.05E-10
Vegetation Contact						
Hiker	4.36E-10	4.65E-10	6.97E-10	1.16E-09	1.39E-11	5.81E-12
Picker	1.64E-07	1.75E-07	2.63E-07	4.38E-07	5.26E-09	2.19E-09
Drinking Water	1.09E-07	1.09E-07	1.05E-07	1.75E-07	4.37E-08	8.74E-08
Eating Berries	1.50E-07	1.50E-07	1.44E-07	2.40E-07	6.00E-08	1.20E-07
Eating Vegets.	3.00E-07	3.00E-07	2.88E-07	4.80E-07	1.20E-07	2.40E-07
Eating Deer	1.84E-08	1.84E-08	1.80E-08	2.99E-08	7.22E-09	1.44E-08
Eating Fish	4.37E-08	4.37E-08	4.19E-08	6.99E-08	1.75E-08	3.49E-08
<u>Combined Routes of Exposure</u>						
Hiker	1.40E-07	1.42E-07	1.54E-07	2.57E-07	4.47E-08	8.78E-08
Berry Picker	4.54E-07	4.67E-07	5.60E-07	9.34E-07	1.10E-07	2.10E-07
Hunter	2.06E-07	2.08E-07	2.20E-07	3.66E-07	6.98E-08	1.38E-07
Fisherman	1.84E-07	1.86E-07	1.96E-07	3.27E-07	6.21E-08	1.23E-07
Resident	4.40E-07	4.42E-07	4.42E-07	7.37E-07	1.65E-07	3.28E-07

Table B-30
Lifetime Doses for Exposed Public (mg/kg/day)
Large Right of Way, Worst Case

Exposures per Lifetime		Average Daily Dose for Exclusive Use of						
		2,4-D	2,4-DP	Asulam	Bromacil	Picloram	Amitrole	Glyphosate
For a Single Exposure								
Dermal, Spray	1	5.77E-09	7.51E-09	1.17E-08	2.35E-08	2.25E-10	1.88E-10	1.17E-08
Vegetation Contact								
Hiker	1	8.28E-11	1.08E-10	1.68E-10	3.37E-10	3.23E-12	2.69E-12	1.68E-10
Picker	1	1.49E-08	1.94E-08	3.02E-08	6.05E-08	5.81E-10	4.84E-10	3.02E-08
Drinking Water	1	7.99E-09	9.74E-09	9.74E-09	1.95E-08	3.90E-09	1.56E-08	9.74E-09
Eating Berries	1	1.24E-08	1.52E-08	1.52E-08	3.03E-08	6.07E-09	2.43E-08	1.52E-08
Eating Vegets.	1	2.49E-08	3.03E-08	3.03E-08	6.07E-08	1.21E-08	4.85E-08	3.03E-08
Eating Deer	1	1.55E-09	1.89E-09	1.92E-09	3.84E-09	7.39E-10	2.95E-09	1.92E-09
Eating Fish	1	3.20E-09	3.90E-09	3.90E-09	7.79E-09	1.56E-09	6.23E-09	3.90E-09
Combined Routes of Exposure								
Hiker	1	1.38E-08	1.74E-08	2.16E-08	4.33E-08	4.13E-09	1.58E-08	2.16E-08
Berry Picker	1	4.11E-08	5.18E-08	6.69E-08	1.34E-07	1.08E-08	4.05E-08	6.69E-08
Hunter	1	1.96E-08	2.44E-08	2.90E-08	5.79E-08	6.80E-09	2.64E-08	2.90E-08
Fisherman	1	1.70E-08	2.13E-08	2.55E-08	5.11E-08	5.68E-09	2.20E-08	2.55E-08
Resident	1	3.87E-08	4.77E-08	5.20E-08	1.04E-07	1.63E-08	6.43E-08	5.20E-08
For 30 Exposures								
Dermal, Spray	30	1.73E-07	2.25E-07	3.52E-07	7.04E-07	6.76E-09	5.63E-09	3.52E-07
Vegetation Contact								
Hiker	30	2.48E-09	3.23E-09	5.05E-09	1.01E-08	9.70E-11	8.08E-11	5.05E-09
Picker	30	4.46E-07	5.81E-07	9.07E-07	1.81E-06	1.74E-08	1.45E-08	9.07E-07
Drinking Water	30	2.40E-07	2.92E-07	2.92E-07	5.85E-07	1.17E-07	4.68E-07	2.92E-07
Eating Berries	30	3.73E-07	4.55E-07	4.55E-07	9.10E-07	1.82E-07	7.28E-07	4.55E-07
Eating Vegets.	30	7.46E-07	9.10E-07	9.10E-07	1.82E-06	3.64E-07	1.46E-06	9.10E-07
Eating Deer	30	4.65E-08	5.68E-08	5.76E-08	1.15E-07	2.22E-08	8.85E-08	5.76E-08
Eating Fish	30	9.59E-08	1.17E-07	1.17E-07	2.34E-07	4.68E-08	1.87E-07	1.17E-07
Combined Routes of Exposure								
Hiker	30	4.15E-07	5.21E-07	6.49E-07	1.30E-06	1.24E-07	4.73E-07	6.49E-07
Berry Picker	30	1.23E-06	1.55E-06	2.01E-06	4.01E-06	3.23E-07	1.22E-06	2.01E-06
Hunter	30	5.89E-07	7.33E-07	8.69E-07	1.74E-06	2.04E-07	7.93E-07	8.69E-07
Fisherman	30	5.11E-07	6.38E-07	7.66E-07	1.53E-06	1.71E-07	6.60E-07	7.66E-07
Resident	30	1.16E-06	1.43E-06	1.56E-06	3.12E-06	4.88E-07	1.93E-06	1.56E-06

Table B-31
Lifetime Doses for Exposed Public (mg/kg/day)
Accidental Spraying

Exposures per Lifetime		Average Daily Dose for Exclusive Use of					Amitrole	Glyphosate
		2,4-D	2,4-DP	Asulam	Bromacil	Picloram		
<u>For a Single Exposure</u>								
Dermal, Spray	1	4.02E-06	5.22E-06	8.16E-06	1.63E-05	3.92E-07	1.31E-07	8.16E-06
Vegetation Contact								
Hiker	1	5.76E-08	7.49E-08	1.17E-07	2.34E-07	5.62E-09	1.87E-09	1.17E-07
Picker	1	1.04E-05	1.35E-05	2.10E-05	4.21E-05	1.01E-06	3.37E-07	2.10E-05
Drinking Water	1	2.35E-06	2.87E-06	2.87E-06	5.74E-06	2.87E-06	4.59E-06	2.87E-06
Eating Berries	1	1.87E-06	2.28E-06	2.28E-06	4.56E-06	2.28E-06	3.65E-06	2.28E-06
Eating Vegets.	1	3.88E-06	4.73E-06	4.73E-06	9.47E-06	4.73E-06	7.57E-06	4.73E-06
Eating Deer	1	4.02E-07	4.92E-07	5.11E-07	1.02E-06	4.60E-07	7.34E-07	5.11E-07
Eating Fish	1	9.42E-07	1.15E-06	1.15E-06	2.30E-06	1.15E-06	1.84E-06	1.15E-06
<u>Combined Routes of Exposure</u>								
Hiker	1	6.43E-06	8.17E-06	1.12E-05	2.23E-05	3.27E-06	4.73E-06	1.12E-05
Berry Picker	1	1.86E-05	2.38E-05	3.44E-05	6.87E-05	6.55E-06	8.71E-06	3.44E-05
Hunter	1	9.35E-06	1.18E-05	1.49E-05	2.98E-05	6.58E-06	1.00E-05	1.49E-05
Fisherman	1	7.37E-06	9.32E-06	1.23E-05	2.46E-05	4.42E-06	6.56E-06	1.23E-05
Resident	1	1.03E-05	1.29E-05	1.59E-05	3.18E-05	8.00E-06	1.23E-05	1.59E-05
<u>For 30 Exposures</u>								
Dermal, Spray	30	1.20E-04	1.57E-04	2.45E-04	4.90E-04	1.18E-05	3.92E-06	2.45E-04
Vegetation Contact								
Hiker	30	1.73E-06	2.25E-06	3.51E-06	7.03E-06	1.69E-07	5.62E-08	3.51E-06
Picker	30	3.11E-04	4.04E-04	6.31E-04	1.26E-03	3.03E-05	1.01E-05	6.31E-04
Drinking Water	30	7.06E-05	8.61E-05	8.61E-05	1.72E-04	8.61E-05	1.38E-04	8.61E-05
Eating Berries	30	5.61E-05	6.84E-05	6.84E-05	1.37E-04	6.84E-05	1.09E-04	6.84E-05
Eating Vegets.	30	1.16E-04	1.42E-04	1.42E-04	2.84E-04	1.42E-04	2.27E-04	1.42E-04
Eating Deer	30	1.20E-05	1.48E-05	1.53E-05	3.07E-05	1.38E-05	2.20E-05	1.53E-05
Eating Fish	30	2.83E-05	3.45E-05	3.45E-05	6.89E-05	3.45E-05	5.51E-05	3.45E-05
<u>Combined Routes of Exposure</u>								
Hiker	30	1.93E-04	2.45E-04	3.35E-04	6.69E-04	9.81E-05	1.42E-04	3.35E-04
Berry Picker	30	5.58E-04	7.15E-04	1.03E-03	2.06E-03	1.97E-04	2.61E-04	1.03E-03
Hunter	30	2.81E-04	3.53E-04	4.47E-04	8.94E-04	1.98E-04	3.00E-04	4.47E-04
Fisherman	30	2.21E-04	2.80E-04	3.69E-04	7.38E-04	1.33E-04	1.97E-04	3.69E-04
Resident	30	3.09E-04	3.87E-04	4.77E-04	9.53E-04	2.40E-04	3.69E-04	4.77E-04

Table B-32
Lifetime Doses for Exposure Due to Spills (mg/kg/day)

Exposures per Lifetime		Average Daily Dose for Exclusive Use of							
		2,4-D	2,4-DP	Asulam	Bromacil	Picloram	Amitrole	Glyphosate	Atrazine
For a Single Exposure									
Spills onto Skin									
Concentrate	1	5.64E-03	9.02E-03	9.39E-03	9.39E-03	2.25E-04	4.70E-05	7.05E-03	9.39E-03
Spray Mix	1	5.64E-04	3.76E-04	7.84E-04	4.70E-04	5.64E-05	9.39E-06	1.17E-03	9.39E-04
Spills into Water (1 Liter Drunk)									
Pond, Helo.	1	2.88E-06	1.80E-06	2.41E-06	-----	3.60E-06	2.88E-06	3.60E-06	2.88E-06
Reserv., Helo.	1	9.01E-08	5.63E-08	7.52E-08	-----	1.13E-07	9.01E-08	1.13E-07	9.01E-08
Pond, Truck	1	5.77E-05	3.60E-05	4.82E-05	2.88E-05	7.21E-05	5.77E-05	7.21E-05	5.77E-05
Reserv., Truck	1	1.80E-06	1.13E-06	1.50E-06	9.00E-07	2.25E-06	1.80E-06	2.25E-06	1.80E-06

Appendix D
Human Health Risk
Assessment
(Quantitative)

Attachment C

Table C-1

Margins of Safety for Workers Using Amitrole

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (4080.0)	SYSTEMIC NOEL (0.03)	REPRODUCTIVE NOEL (5.00)
<u>Routine-Realistic Exposures</u>				
PILOT	0.0004	1000000+	62	12000
MIXER/LOADER	0.0006	1000000+	43	8700
SUPERVISOR	0.0001	1000000+	410	81000
OBSERVER	0.0000	1000000+	1900	380000
BACKPACK	0.0033	1000000+	7.6	1500
R-O-W SPRAYER	0.0001	1000000+	360	73000
R-O-W MIX/L	0.0001	1000000+	360	72000
R-O-W AP/M/L	0.0001	1000000+	260	52000
HACK & SQUIRT	0.0006	1000000+	45	9000
INJECTION BAR	0.0002	1000000+	120	24000
<u>Routine-Worst Case Exposures</u>				
PILOT	0.0067	610000	3.7	750
MIXER/LOADER	0.0085	480000	2.9	590
SUPERVISOR	0.0012	1000000+	22	4300
OBSERVER	0.0002	1000000+	120	24000
BACKPACK	0.0310	130000	-1.2	160
R-O-W SPRAYER	0.0042	960000	5.9	1200
R-O-W MIX/L	0.0024	1000000+	10	2000
R-O-W AP/M/L	0.0029	1000000+	8.7	1700
HACK & SQUIRT	0.0063	650000	4.0	790
INJECTION BAR	0.0017	1000000+	15	3000

^aThe plus sign (+) means "greater than."

Table C-2

Margins of Safety for Workers Using Asulam

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (4000.0)	SYSTEMIC NOEL (50.00)	REPRODUCTIVE NOEL (50.00)
<u>Routine-Realistic Exposures</u>				
PILOT	0.0483	83000	1000	1000
MIXER/LOADER	0.0693	58000	720	720
SUPERVISOR	0.0074	540000	6800	6800
OBSERVER	0.0016	1000000+	32000	32000
BACKPACK	0.1978	20000	250	250
R-O-W SPRAYER	0.0082	490000	6100	6100
R-O-W MIX/L	0.0084	480000	6000	6000
R-O-W AP/M/L	0.0116	350000	4300	4300
HACK & SQUIRT	---	---	---	---
INJECTION BAR	---	---	---	---
<u>Routine-Worst Case Exposures</u>				
PILOT	0.5591	7200	89	89
MIXER/LOADER	0.7128	5600	70	70
SUPERVISOR	0.0969	41000	520	520
OBSERVER	0.0173	230000	2900	2900
BACKPACK	2.0693	1900	24	24
R-O-W SPRAYER	0.2652	15000	190	190
R-O-W MIX/L	0.1530	26000	330	330
R-O-W AP/M/L	0.1787	22000	280	280
HACK & SQUIRT	---	---	---	---
INJECTION BAR	---	---	---	---

^aThe plus sign (+) means "greater than."

Table C-3

Margins of Safety for Workers Using Atrazine

MARGIN OF SAFETY RELATIVE TO			
EXPOSURE (MG/KG/DAY)	LD50 (1869.0)	SYSTEMIC NOEL (3.70)	REPRODUCTIVE NOEL (100.00)

Routine-Realistic Exposures

MARGIN OF SAFETY RELATIVE TO:			
EXPOSURE (MG/KG/DAY)	LD50 (1869.0)	SYSTEMIC NOEL (3.70)	REPRODUCTIVE NOEL (100.00)

Routine-Realistic Exposures

PILOT	0.0755	25000	49	1300
MIXER/LOADER	0.1083	17000	34	920
SUPERVISOR	0.0116	160000	320	8700
OBSERVER	0.0025	760000	1500	41000
BACKPACK	0.4946	3800	7.5	200
R-O-W SPRAYER	0.0103	180000	360	9700
R-O-W MIX/L	0.0105	180000	350	9500
R-O-W AP/M/L	0.0145	130000	260	6900
HACK & SQUIRT	---	---	---	---
INJECTION BAR	---	---	---	---

Routine-Worst Case Exposures

PILOT	0.6696	2800	5.5	150
MIXER/LOADER	0.8536	2200	4.3	120
SUPERVISOR	0.1160	16000	32	860
OBSERVER	0.0207	90000	180	4800
BACKPACK	2.4782	750	1.5	40
R-O-W SPRAYER	0.4509	4100	8.2	220
R-O-W MIX/L	0.2601	7200	14	380
R-O-W AP/M/L	0.3038	6200	12	330
HACK & SQUIRT	---	---	---	---
INJECTION BAR	---	---	---	---

Table C-4

Margins of Safety for Workers Using Bromacil

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (3998.0)	SYSTEMIC NOEL (6.25)	REPRODUCTIVE NOEL (7.92)
<u>Routine-Realistic Exposures</u>				
PILOT	---	---	---	---
MIXER/LOADER	---	---	---	---
SUPERVISOR	---	---	---	---
OBSERVER	---	---	---	---
BACKPACK	0.6595	6100	9.5	12
R-O-W SPRAYER	0.0137	290000	460	580
R-O-W MIX/L	0.0140	290000	450	570
R-O-W AP/M/L	0.0193	210000	320	410
HACK & SQUIRT	0.1113	36000	56	71
INJECTION BAR	0.0424	94000	150	190
<u>Routine-Worst Case Exposures</u>				
PILOT	---	---	---	---
MIXER/LOADER	---	---	---	---
SUPERVISOR	---	---	---	---
OBSERVER	---	---	---	---
BACKPACK	6.1955	650	1.0	1.3
R-O-W SPRAYER	0.5305	7500	12	15
R-O-W MIX/L	0.3060	13000	20	26
R-O-W AP/M/L	0.3574	11000	17	22
HACK & SQUIRT	1.2617	3200	5.0	6.3
INJECTION BAR	0.3373	12000	19	23

Table C-5

Margins of Safety for Workers Using 2,4-D

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE	LD50	SYSTEMIC	REPRODUCTIVE
	(MG/KG/DAY)	(375.0)	NOEL (1.00)	NOEL (5.00)
<u>Routine-Realistic Exposures</u>				
PILOT	0.0302	12000	33	166
MIXER/LOADER	0.0433	8700	23	116
SUPERVISOR	0.0046	81000	220	1100
OBSERVER	0.0010	380000	1000	5200
BACKPACK	0.1978	1900	5.1	26
R-O-W SPRAYER	0.0051	73000	190	980
R-O-W MIX/L	0.0052	72000	190	960
R-O-W AP/M/L	0.0072	52000	140	700
HACK & SQUIRT	0.0668	5600	15	74
INJECTION BAR	0.0254	15000	39	200
<u>Routine-Worst Case Exposures</u>				
PILOT	0.4018	930	2.5	12
MIXER/LOADER	0.5122	730	2.0	10
SUPERVISOR	0.0696	5400	14	72
OBSERVER	0.0124	30000	81	500
BACKPACK	1.4869	250	-1.5	3.4
R-O-W SPRAYER	0.1305	2900	7.7	38
R-O-W MIX/L	0.0753	5000	13	66
R-O-W AP/M/L	0.0879	4300	11	56
HACK & SQUIRT	0.7570	500	1.3	6.6
INJECTION BAR	0.2024	1900	4.9	24

Table C-6

Margins of Safety for Workers Using 2,4-DP

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (532.0)	SYSTEMIC NOEL (5.00)	REPRODUCTIVE NOEL (6.25)
<u>Routine-Realistic Exposures</u>				
PILOT	0.0258	21000	190	240
MIXER/LOADER	0.0370	14000	140	170
SUPERVISOR	0.0039	130000	1300	1600
OBSERVER	0.0008	640000	6000	7500
BACKPACK	0.2110	2500	24	30
R-O-W SPRAYER	0.0055	97000	910	1100
R-O-W MIX/L	0.0056	95000	890	1100
R-O-W AP/M/L	0.0077	69000	650	810
HACK & SQUIRT	0.1069	5000	47	58
INJECTION BAR	0.0407	13000	120	150
<u>Routine-Worst Case Exposures</u>				
PILOT	0.2678	2000	19	23
MIXER/LOADER	0.3414	1600	15	18
SUPERVISOR	0.0464	11000	110	130
OBSERVER	0.0083	64000	600	760
BACKPACK	1.7050	310	2.9	3.7
R-O-W SPRAYER	0.1698	3100	29	37
R-O-W MIX/L	0.0979	5400	51	64
R-O-W AP/M/L	0.1144	4700	44	55
HACK & SQUIRT	1.2112	440	4.1	5.2
INJECTION BAR	0.3238	1600	15	19

Table C-7

Margins of Safety for Workers Using Dalapon

MARGIN OF SAFETY RELATIVE TO				
EXPOSURE (MG/KG/DAY)	LD50 (7577.0)	SYSTEMIC NOEL (15.00)	REPRODUCTIVE NOEL (300.00)	
<u>Routine-Realistic Exposures</u>				
EXPOSURE (MG/KG/DAY)	LD50 (7577.0)	SYSTEMIC NOEL (15.00)	REPRODUCTIVE NOEL (300.00)	
PILOT	0.0806	94000	190	3700
MIXER/LOADER	0.1156	66000	130	2600
SUPERVISOR	0.0123	620000	1200	24000
OBSERVER	0.0026	1000000+	5700	110000
BACKPACK	0.6595	11000	23	450
R-O-W SPRAYER	0.0137	550000	1100	22000
R-O-W MIX/L	0.0140	540000	1100	21000
R-O-W AP/M/L	0.0193	390000	780	16000
HACK & SQUIRT	---	---	---	---
INJECTION BAR	---	---	---	---
<u>Routine-Worst Case Exposures</u>				
PILOT	1.6740	4500	9.0	180
MIXER/LOADER	2.1340	3600	7.0	140
SUPERVISOR	0.2900	26000	52	1000
OBSERVER	0.0517	150000	290	5800
BACKPACK	7.4346	1000	2.0	40
R-O-W SPRAYER	0.5305	14000	28	570
R-O-W MIX/L	0.3060	25000	49	980
R-O-W AP/M/L	0.3574	21000	42	840
HACK & SQUIRT	---	---	---	---
INJECTION BAR	---	---	---	---

^aThe plus sign (+) means "greater than."

Table C-8

Margins of Safety for Workers Using Dicamba

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (757.0)	SYSTEMIC NOEL (25.00)	REPRODUCTIVE NOEL (2.50)
<u>Routine-Realistic Exposures</u>				
PILOT	0.0101	75000	2500	250
MIXER/LOADER	0.0144	52000	1700	170
SUPERVISOR	0.0015	490000	16000	1600
OBSERVER	0.0003	1000000+	77000	7700
BACKPACK	0.0412	18000	610	61
R-O-W SPRAYER	0.0017	440000	15000	1500
R-O-W MIX/L	0.0017	430000	14000	1400
R-O-W AP/M/L	0.0024	310000	10000	1000
HACK & SQUIRT	0.0557	14000	450	45
INJECTION BAR	0.0212	36000	1200	120
<u>Routine-Worst Case Exposures</u>				
PILOT	0.3348	2300	75	7.5
MIXER/LOADER	0.4268	1800	59	5.9
SUPERVISOR	0.0580	13000	430	43
OBSERVER	0.0103	73000	2400	240
BACKPACK	1.2391	610	20	2.0
R-O-W SPRAYER	0.0955	7900	260	26
R-O-W MIX/L	0.0551	14000	450	45
R-O-W AP/M/L	0.0643	12000	390	39
HACK & SQUIRT	0.6308	1200	40	4.0
INJECTION BAR	0.1686	4500	150	15

^aThe plus sign (+) means "greater than."

Table C-9

Margins of Safety for Workers Using Diuron

MARGIN OF SAFETY RELATIVE TO			
EXPOSURE (MG/KG/DAY)	LD50 (3750.0)	SYSTEMIC NOEL (0.63)	REPRODUCTIVE NOEL (6.25)

Routine-Realistic Exposures

MARGIN OF SAFETY RELATIVE TO:			
EXPOSURE (MG/KG/DAY)	LD50 (3750.0)	SYSTEMIC NOEL (0.63)	REPRODUCTIVE NOEL (6.25)

Routine-Realistic Exposures

PILOT	---	---	---	---
MIXER/LOADER	---	---	---	---
SUPERVISOR	---	---	---	---
OBSERVER	---	---	---	---
BACKPACK	0.6595	5700	-1.1	9.5
R-O-W SPRAYER	0.0137	270000	46	460
R-O-W MIX/L	0.0140	270000	45	450
R-O-W AP/M/L	0.0193	190000	32	320
HACK & SQUIRT	0.1113	34000	5.6	56
INJECTION BAR	0.0424	89000	15	150

Routine-Worst Case Exposures

PILOT	---	---	---	---
MIXER/LOADER	---	---	---	---
SUPERVISOR	---	---	---	---
OBSERVER	---	---	---	---
BACKPACK	3.7173	1000	-5.9	1.7
R-O-W SPRAYER	0.8488	4400	-1.4	7.4
R-O-W MIX/L	0.4896	7700	1.3	13
R-O-W AP/M/L	0.5719	6600	1.1	11
HACK & SQUIRT	1.2617	3000	-2.0	5.0
INJECTION BAR	0.3373	11000	1.9	19

Table C-10

Margins of Safety for Workers Using Fosamine

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (24400.0)	SYSTEMIC NOEL (25.00)	REPRODUCTIVE NOEL (500.00)
<u>Routine-Realistic Exposures</u>				
PILOT	0.0604	400000	410	8000
MIXER/LOADER	0.0867	280000	290	5800
SUPERVISOR	0.0092	1000000+	2700	54000
OBSERVER	0.0020	1000000+	13000	250000
BACKPACK	0.4946	49000	51	1000
R-O-W SPRAYER	0.0137	1000000+	1800	37000
R-O-W MIX/L	0.0140	1000000+	1800	36000
R-O-W AP/M/L	0.0193	1000000+	1300	26000
HACK & SQUIRT	0.1113	220000	220	4500
INJECTION BAR	0.0424	580000	590	12000
<u>Routine-Worst Case Exposures</u>				
PILOT	2.0088	12000	12	250
MIXER/LOADER	2.5608	9500	9.8	190
SUPERVISOR	0.3480	70000	72	2500
OBSERVER	0.0620	390000	400	8000
BACKPACK	7.1248	3400	3.5	73
R-O-W SPRAYER	0.5676	43000	44	870
R-O-W MIX/L	0.3274	75000	76	1500
R-O-W AP/M/L	0.3824	64000	65	1300
HACK & SQUIRT	1.2617	19000	20	390
INJECTION BAR	0.3373	72000	74	1500

^aThe plus sign (+) means "greater than."

Table C-11

Margins of Safety for Workers Using Glyphosate

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (4320.0)	SYSTEMIC NOEL (31.0)	REPRODUCTIVE NOEL (10.00)
<u>Routine-Realistic Exposures</u>				
PILOT	0.0403	110000	770	250
MIXER/LOADER	0.0578	75000	540	170
SUPERVISOR	0.0062	700000	5000	1600
OBSERVER	0.0013	1000000+	24000	7700
BACKPACK	0.2473	17000	130	40
R-O-W SPRAYER	0.0069	630000	4600	1500
R-O-W MIX/L	0.0070	620000	4500	1400
R-O-W AP/M/L	0.0096	450000	3200	1000
HACK & SQUIRT	---	---	---	---
INJECTION BAR	---	---	---	---
<u>Routine-Worst Case Exposures</u>				
PILOT	0.8370	5200	37	12
MIXER/LOADER	1.0670	4000	29	9.4
SUPERVISOR	0.1450	30000	218	69
OBSERVER	0.0258	170000	1200	390
BACKPACK	3.0978	1400	10	3.2
R-O-W SPRAYER	0.2652	16000	120	38
R-O-W MIX/L	0.1530	28000	210	65
R-O-W AP/M/L	0.1787	24000	180	56
HACK & SQUIRT	---	---	---	---
INJECTION BAR	---	---	---	---

^aThe plus sign (+) means "greater than."

Table C-12

Margins of Safety for Workers Using Hexazinone

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (1690.0)	SYSTEMIC NOEL (10.00)	REPRODUCTIVE NOEL (125.00)
<u>Routine-Realistic Exposures</u>				
PILOT	0.0504	34000	200	2500
MIXER/LOADER	0.0722	23000	140	1700
SUPERVISOR	0.0077	220000	1300	16000
OBSERVER	0.0016	1000000	6100	77000
BACKPACK	0.1846	9200	54	680
R-O-W SPRAYER	0.0086	200000	1200	15000
R-O-W MIX/L	0.0087	190000	1100	14000
R-O-W AP/M/L	0.0120	140000	830	10000
HACK & SQUIRT	---	---	---	---
INJECTION BAR	---	---	---	---
<u>Routine-Worst Case Exposures</u>				
PILOT	0.5022	3400	20	250
MIXER/LOADER	0.6402	2600	16	200
SUPERVISOR	0.0870	19000	110	1400
OBSERVER	0.0155	110000	650	8100
BACKPACK	1.8587	910	5.4	67
R-O-W SPRAYER	0.3183	5300	31	390
R-O-W MIX/L	0.1836	9200	54	680
R-O-W AP/M/L	0.2145	7900	47	580
HACK & SQUIRT	---	---	---	---
INJECTION BAR	---	---	---	---

Table C-13

Margins of Safety for Workers Using Picloram

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (8200.0)	SYSTEMIC NOEL (7.00)	REPRODUCTIVE NOEL (50.00)
<u>Routine-Realistic Exposures</u>				
PILOT	0.0010	1000000+	7200	52000
MIXER/LOADER	0.0014	1000000+	5000	36000
SUPERVISOR	0.0001	1000000+	47000	340000
OBSERVER	0.0000	1000000+	220000	1000000+
BACKPACK	0.0079	1000000	880	6300
R-O-W SPRAYER	0.0002	1000000+	43000	300000
R-O-W MIX/L	0.0002	1000000+	42000	300000
R-O-W AP/M/L	0.0002	1000000+	30000	220000
HACK & SQUIRT	0.0027	1000000+	2600	19000
INJECTION BAR	0.0010	1000000+	6900	49000
<u>Routine-Worst Case Exposures</u>				
PILOT	0.0402	200000	170	1200
MIXER/LOADER	0.0512	160000	140	980
SUPERVISOR	0.0070	1000000+	1000	7200
OBSERVER	0.0012	1000000+	5600	40000
BACKPACK	0.1190	69000	59	420
R-O-W SPRAYER	0.0051	1000000+	1400	9800
R-O-W MIX/L	0.0029	1000000+	2400	17000
R-O-W AP/M/L	0.0034	1000000+	2000	15000
HACK & SQUIRT	0.0303	270000	230	1700
INJECTION BAR	0.0081	1000000	860	6200

^aThe plus sign (+) means "greater than."

Table C-14

Margins of Safety for Workers Using Simazine

MARGIN OF SAFETY RELATIVE TO				
EXPOSURE (MG/KG/DAY)	LD50 (5000.0)	SYSTEMIC NOEL (5.00)	REPRODUCTIVE NOEL (5.00)	
<u>Routine-Realistic Exposures</u>				
MARGIN OF SAFETY RELATIVE TO:				
EXPOSURE (MG/KG/DAY)	LD50 (5000.0)	SYSTEMIC NOEL (5.00)	REPRODUCTIVE NOEL (5.00)	
<u>Routine-Realistic Exposures</u>				
PILOT	0.0806	62000	62	62
MIXER/LOADER	0.1156	43000	43	43
SUPERVISOR	0.0123	410000	410	410
OBSERVER	0.0026	1000000+	1900	1900
BACKPACK	0.3297	15000	15	15
R-O-W SPRAYER	0.0069	730000	730	730
R-O-W MIX/L	0.0070	720000	720	720
R-O-W AP/M/L	0.0096	520000	520	520
HACK & SQUIRT	---	---	---	---
INJECTION BAR	---	---	---	---
<u>Routine-Worst Case Exposures</u>				
PILOT	0.8370	6000	6.0	6.0
MIXER/LOADER	1.0670	4700	4.7	4.7
SUPERVISOR	0.1450	34000	34	34
OBSERVER	0.0258	190000	190	190
BACKPACK	2.8499	1800	1.8	1.8
R-O-W SPRAYER	0.2440	20000	20	20
R-O-W MIX/L	0.1408	36000	36	36
R-O-W AP/M/L	0.1644	30000	30	30
HACK & SQUIRT	---	---	---	---
INJECTION BAR	---	---	---	---

^aThe plus sign (+) means "greater than."

Table C-15

Margins of Safety for Workers Using Tebuthiuron

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (644.0)	SYSTEMIC NOEL (12.50)	REPRODUCTIVE NOEL (90.00)
<u>Routine-Realistic Exposures</u>				
PILOT	0.0201	32000	620	4500
MIXER/LOADER	0.0289	22000	430	3100
SUPERVISOR	0.0031	210000	4100	29000
OBSERVER	0.0007	990000	19000	140000
BACKPACK	0.2473	2600	51	360
R-O-W SPRAYER	0.0075	85000	1700	12000
R-O-W MIX/L	0.0077	84000	1600	12000
R-O-W AP/M/L	0.0106	61000	1200	8500
HACK & SQUIRT	---	---	---	---
INJECTION BAR	---	---	---	---
<u>Routine-Worst Case Exposures</u>				
PILOT	1.0044	640	12	90
MIXER/LOADER	1.2804	500	9.8	70
SUPERVISOR	0.1740	3700	72	520
OBSERVER	0.0310	21000	400	2900
BACKPACK	3.7173	170	3.4	24
R-O-W SPRAYER	0.2440	2600	51	370
R-O-W MIX/L	0.1408	4600	89	640
R-O-W AP/M/L	0.1644	3900	76	550
HACK & SQUIRT	---	---	---	---
INJECTION BAR	---	---	---	---

Table C-16

Margins of Safety for Workers Using Triclopyr

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (630.0)	SYSTEMIC NOEL (2.50)	REPRODUCTIVE NOEL (10.00)
<u>Routine-Realistic Exposures</u>				
PILOT	0.0403	16000	62	250
MIXER/LOADER	0.0578	11000	43	170
SUPERVISOR	0.0062	100000	410	1600
OBSERVER	0.0013	480000	1900	7700
BACKPACK	0.3297	1900	7.6	30
R-O-W SPRAYER	0.0069	92000	360	1500
R-O-W MIX/L	0.0070	90000	360	1400
R-O-W AP/M/L	0.0096	65000	260	1000
HACK & SQUIRT	0.1113	5700	22	90
INJECTION BAR	0.0424	15000	59	240
<u>Routine-Worst Case Exposures</u>				
PILOT	1.3392	470	1.9	7.5
MIXER/LOADER	1.7072	370	1.5	5.9
SUPERVISOR	0.2320	2700	11	43
OBSERVER	0.0413	15000	60	240
BACKPACK	4.9564	130	-2.0	2.0
R-O-W SPRAYER	0.4244	1500	5.9	24
R-O-W MIX/L	0.2448	2600	10	41
R-O-W AP/M/L	0.2859	2200	8.7	35
HACK & SQUIRT	1.2617	500	2.0	7.9
INJECTION BAR	0.3373	1900	7.4	30

Table C-17

Margins of Safety for Workers Wearing
Protective Clothing and Using Amitrole

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (4080.0)	SYSTEMIC NOEL (0.03)	REPRODUCTIVE NOEL (5.00)
<u>Routine-Realistic Exposures</u>				
PILOT	0.0002	1000000+	150	30000
MIXER/LOADER	0.0004	1000000+	59	12000
SUPERVISOR	0.0000	1000000+	970	190000
OBSERVER	0.0000	1000000+	4600	910000
BACKPACK	0.0010	1000000+	24	4900
R-O-W SPRAYER	0.0000	1000000+	1200	230000
R-O-W MIX/L	0.0001	1000000+	490	98000
R-O-W AP/M/L	0.0001	1000000+	500	100000
HACK & SQUIRT	0.0002	1000000+	110	21000
INJECTION BAR	0.0001	1000000+	260	52000
<u>Routine-Worst Case Exposures</u>				
PILOT	0.0028	1000000+	8.9	1800
MIXER/LOADER	0.0062	660000	4.0	800
SUPERVISOR	0.0005	1000000+	51	10000
OBSERVER	0.0001	1000000+	290	58000
BACKPACK	0.0097	420000	2.6	520
R-O-W SPRAYER	0.0013	1000000+	19	3800
R-O-W MIX/L	0.0018	1000000+	14	2800
R-O-W AP/M/L	0.0015	1000000+	17	3400
HACK & SQUIRT	0.0027	1000000+	9.3	1900
INJECTION BAR	0.0008	1000000+	33	6500

^aThe plus sign (+) means "greater than."

Table C-18

Margins of Safety for Workers Wearing
Protective Clothing and Using Asulam

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE	LD50	SYSTEMIC	REPRODUCTIVE
	(MG/KG/DAY)	(4000.0)	NOEL (50.00)	NOEL (50.00)
<u>Routine-Realistic Exposures</u>				
PILOT	0.0203	200000	3000	2500
MIXER/LOADER	0.0505	79000	1200	990
SUPERVISOR	0.0031	1000000+	19000	16000
OBSERVER	0.0007	1000000+	91000	76000
BACKPACK	0.0617	65000	970	810
R-O-W SPRAYER	0.0026	1000000+	23000	19000
R-O-W MIX/L	0.0061	650000	9800	8200
R-O-W AP/M/L	0.0060	660000	10000	8300
HACK & SQUIRT	---	---	---	---
INJECTION BAR	---	---	---	---
<u>Routine-Worst Case Exposures</u>				
PILOT	0.2343	17000	260	210
MIXER/LOADER	0.5196	7700	120	96
SUPERVISOR	0.0406	99000	1500	1200
OBSERVER	0.0072	550000	8300	6900
BACKPACK	0.6456	6200	93	77
R-O-W SPRAYER	0.0828	48000	730	600
R-O-W MIX/L	0.1115	36000	540	450
R-O-W AP/M/L	0.0930	43000	650	540
HACK & SQUIRT	---	---	---	---
INJECTION BAR	---	---	---	---

^aThe plus sign (+) means "greater than."

Table C-19

Margins of Safety for Workers Wearing
Protective Clothing and Using Atrazine

MARGIN OF SAFETY RELATIVE TO				
EXPOSURE (MG/KG/DAY)	LD50 (1869.0)	SYSTEMIC NOEL (3.70)	REPRODUCTIVE NOEL (100.00)	
<u>Routine-Realistic Exposures</u>				
PILOT	0.0317	59000	120	3200
MIXER/LOADER	0.0790	24000	47	1300
SUPERVISOR	0.0048	390000	760	21000
OBSERVER	0.0010	1000000+	3600	97000
BACKPACK	0.1543	12000	24	650
R-O-W SPRAYER	0.0032	580000	1200	31000
R-O-W MIX/L	0.0076	240000	480	13000
R-O-W AP/M/L	0.0075	250000	490	13000
HACK & SQUIRT	---	---	---	---
INJECTION BAR	---	---	---	---
<u>Routine-Worst Case Exposures</u>				
PILOT	0.2806	6700	13	360
MIXER/LOADER	0.6223	3000	5.9	160
SUPERVISOR	0.0486	38000	76	2100
OBSERVER	0.0087	220000	430	12000
BACKPACK	0.7732	2400	4.8	130
R-O-W SPRAYER	0.1407	13000	26	710
R-O-W MIX/L	0.1896	9900	20	530
R-O-W AP/M/L	0.1581	12000	23	630
HACK & SQUIRT	---	---	---	---
INJECTION BAR	---	---	---	---

^aThe plus sign (+) means "greater than."

Table C-20

Margins of Safety for Workers Wearing
Protective Clothing and Using Bromacil

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (3998.0)	SYSTEMIC NOEL (6.25)	REPRODUCTIVE NOEL (7.92)
<u>Routine-Realistic Exposures</u>				
PILOT	----	----	----	----
MIXER/LOADER	----	----	----	----
SUPERVISOR	----	----	----	----
OBSERVER	----	----	----	----
BACKPACK	0.2057	19000	30	38
R-O-W SPRAYER	0.0043	930000	1500	1900
R-O-W MIX/L	0.0102	390000	610	780
R-O-W AP/M/L	0.0100	400000	620	790
HACK & SQUIRT	0.0472	85000	130	170
INJECTION BAR	0.0192	210000	330	410
<u>Routine-Worst Case Exposures</u>				
PILOT	----	----	----	----
MIXER/LOADER	----	----	----	----
SUPERVISOR	----	----	----	----
OBSERVER	----	----	----	----
BACKPACK	1.9330	2100	3.2	4.1
R-O-W SPRAYER	0.1655	24000	38	48
R-O-W MIX/L	0.2231	18000	28	36
R-O-W AP/M/L	0.1860	21000	34	43
HACK & SQUIRT	0.5350	7500	12	15
INJECTION BAR	0.1528	26000	41	52

Table C-21

Margins of Safety for Workers Wearing
Protective Clothing and Using 2,4-D

MARGIN OF SAFETY RELATIVE TO				
EXPOSURE (MG/KG/DAY)	LD50 (375.0)	SYSTEMIC NOEL (1.00)	REPRODUCTIVE NOEL (5.00)	
<u>Routine-Realistic Exposures</u>				
PILOT	0.0127	30000	79	400
MIXER/LOADER	0.0316	12000	32	160
SUPERVISOR	0.0019	190000	520	2600
OBSERVER	0.0004	910000	2400	12000
BACKPACK	0.0617	6100	16	82
R-O-W SPRAYER	0.0016	230000	620	3200
R-O-W MIX/L	0.0038	98000	260	1300
R-O-W AP/M/L	0.0038	100000	270	1300
HACK & SQUIRT	0.0283	13000	35	180
INJECTION BAR	0.0115	33000	87	440
<u>Routine-Worst Case Exposures</u>				
PILOT	0.1683	2200	5.9	30
MIXER/LOADER	0.3734	1000	2.7	13
SUPERVISOR	0.0292	13000	34	170
OBSERVER	0.0052	72000	190	960
BACKPACK	0.4639	810	2.2	11
R-O-W SPRAYER	0.0407	9200	25	120
R-O-W MIX/L	0.0549	6800	18	92
R-O-W AP/M/L	0.0458	8200	22	110
HACK & SQUIRT	0.3210	1200	3.1	16
INJECTION BAR	0.0917	4100	11	54

Table C-22

Margins of Safety for Workers Wearing
Protective Clothing and Using 2,4-DP

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (532.0)	SYSTEMIC NOEL (5.00)	REPRODUCTIVE NOEL (6.25)
<u>Routine-Realistic Exposures</u>				
PILOT	0.0108	49000	460	580
MIXER/LOADER	0.0270	20000	190	230
SUPERVISOR	0.0017	320000	3000	3800
OBSERVER	0.0004	1000000+	14000	18000
BACKPACK	0.0658	8100	76	95
R-O-W SPRAYER	0.0017	310000	2900	3700
R-O-W MIX/L	0.0041	130000	1200	1500
R-O-W AP/M/L	0.0040	130000	1200	1600
HACK & SQUIRT	0.0453	12000	110	140
INJECTION BAR	0.0184	29000	270	340
<u>Routine-Worst Case Exposures</u>				
PILOT	0.1122	4700	45	56
MIXER/LOADER	0.2489	2100	20	25
SUPERVISOR	0.0194	27000	260	320
OBSERVER	0.0035	150000	1400	1800
BACKPACK	0.5320	1000	9.4	12
R-O-W SPRAYER	0.0530	10000	94	120
R-O-W MIX/L	0.0714	7500	70	88
R-O-W AP/M/L	0.0595	8900	84	100
HACK & SQUIRT	0.5136	1000	9.7	12
INJECTION BAR	0.1467	3600	34	43

^aThe plus sign (+) means "greater than."

Table C-23

Margins of Safety for Workers Wearing
Protective Clothing and Using Dalapon

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (7577.0)	SYSTEMIC NOEL (15.00)	REPRODUCTIVE NOEL (300.00)
<u>Routine-Realistic Exposures</u>				
PILOT	0.0338	220000	440	8900
MIXER/LOADER	0.0842	90000	180	3600
SUPERVISOR	0.0052	1000000+	2900	58000
OBSERVER	0.0011	1000000+	14000	270000
BACKPACK	0.2057	37000	73	1500
R-O-W SPRAYER	0.0043	1000000+	3500	70000
R-O-W MIX/L	0.0102	740000	1500	29000
R-O-W AP/M/L	0.0100	760000	1500	30000
HACK & SQUIRT	---	---	---	---
INJECTION BAR	---	---	---	---
<u>Routine-Worst Case Exposures</u>				
PILOT	0.7014	11000	21	430
MIXER/LOADER	1.5557	4900	9.6	190
SUPERVISOR	0.1215	62000	120	2500
OBSERVER	0.0216	350000	690	14000
BACKPACK	2.3196	3300	6.5	130
R-O-W SPRAYER	0.1655	46000	91	1800
R-O-W MIX/L	0.2231	34000	67	1300
R-O-W AP/M/L	0.1860	41000	81	1600
HACK & SQUIRT	---	---	---	---
INJECTION BAR	---	---	---	---

^aThe plus sign (+) means "greater than."

Table C-24

Margins of Safety for Workers Wearing
Protective Clothing and Using Dicamba

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (757.0)	SYSTEMIC NOEL (25.00)	REPRODUCTIVE NOEL (2.50)
<u>Routine-Realistic Exposures</u>				
PILOT	0.0042	180000	5900	590
MIXER/LOADER	0.0105	72000	2400	240
SUPERVISOR	0.0006	1000000+	39000	3900
OBSERVER	0.0001	1000000+	180000	18000
BACKPACK	0.0129	59000	1900	190
R-O-W SPRAYER	0.0005	1000000+	47000	4700
R-O-W MIX/L	0.0013	590000	20000	2000
R-O-W AP/M/L	0.0013	600000	20000	2000
HACK & SQUIRT	0.0236	32000	1100	110
INJECTION BAR	0.0096	79000	2600	260
<u>Routine-Worst Case Exposures</u>				
PILOT	0.1403	5400	180	18
MIXER/LOADER	0.3111	2400	80	8.0
SUPERVISOR	0.0243	31000	1000	100
OBSERVER	0.0043	170000	5800	580
BACKPACK	0.3866	2000	65	6.5
R-O-W SPRAYER	0.0298	25000	840	84
R-O-W MIX/L	0.0402	19000	620	62
R-O-W AP/M/L	0.0335	23000	750	75
HACK & SQUIRT	0.2675	2800	93	9.3
INJECTION BAR	0.0764	9900	330	33

^aThe plus sign (+) means "greater than."

Table C-25

Margins of Safety for Workers Wearing
Protective Clothing and Using Diuron

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (3750.0)	SYSTEMIC NOEL (0.63)	REPRODUCTIVE NOEL (6.25)
<u>Routine-Realistic Exposures</u>				
PILOT	---	---	---	---
MIXER/LOADER	---	---	---	---
SUPERVISOR	---	---	---	---
OBSERVER	---	---	---	---
BACKPACK	0.2057	18000	3.0	30
R-O-W SPRAYER	0.0043	880000	150	1500
R-O-W MIX/L	0.0102	370000	61	610
R-O-W AP/M/L	0.0100	370000	62	620
HACK & SQUIRT	0.0472	79000	13	130
INJECTION BAR	0.0192	200000	33	330
<u>Routine-Worst Case Exposures</u>				
PILOT	---	---	---	---
MIXER/LOADER	---	---	---	---
SUPERVISOR	---	---	---	---
OBSERVER	---	---	---	---
BACKPACK	1.1598	3200	-1.9	5.4
R-O-W SPRAYER	0.2648	14000	2.4	24
R-O-W MIX/L	0.3569	11000	1.8	18
R-O-W AP/M/L	0.2977	13000	2.1	21
HACK & SQUIRT	0.5350	7000	1.2	12
INJECTION BAR	0.1528	25000	4.1	41

Table C-26

Margins of Safety for Workers Wearing
Protective Clothing and Using Fosamine

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (24400.0)	SYSTEMIC NOEL (25.00)	REPRODUCTIVE NOEL (500.00)
<u>Routine-Realistic Exposures</u>				
PILOT	0.0253	960000	990	20000
MIXER/LOADER	0.0632	390000	400	8000
SUPERVISOR	0.0039	1000000+	6500	13000
OBSERVER	0.0008	1000000+	30000	610000
BACKPACK	0.1543	160000	160	3300
R-O-W SPRAYER	0.0043	1000000+	5800	120000
R-O-W MIX/L	0.0102	1000000+	2500	49000
R-O-W AP/M/L	0.0100	1000000+	2500	50000
HACK & SQUIRT	0.0472	520000	530	11000
INJECTION BAR	0.0192	1000000+	1300	26000
<u>Routine-Worst Case Exposures</u>				
PILOT	0.8417	29000	30	600
MIXER/LOADER	1.8668	13000	13	270
SUPERVISOR	0.1458	170000	170	3400
OBSERVER	0.0260	940000	960	19000
BACKPACK	2.2229	11000	11	230
R-O-W SPRAYER	0.1771	140000	140	2800
R-O-W MIX/L	0.2387	100000	100	2100
R-O-W AP/M/L	0.1991	120000	130	2500
HACK & SQUIRT	0.5350	46000	47	930
INJECTION BAR	0.1528	160000	160	3300

^aThe plus sign (+) means "greater than."

Table C-27

Margins of Safety for Workers Wearing
Protective Clothing and Using Glyphosate

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (4320.0)	SYSTEMIC NOEL (31.0)	REPRODUCTIVE NOEL (10.00)
<u>Routine-Realistic Exposures</u>				
PILOT	0.0169	260000	1900	590
MIXER/LOADER	0.0421	100000	740	240
SUPERVISOR	0.0026	1000000+	12000	3900
OBSERVER	0.0005	1000000+	57000	18000
BACKPACK	0.0772	56000	410	130
R-O-W SPRAYER	0.0021	1000000+	15000	4700
R-O-W MIX/L	0.0051	850000	6100	2000
R-O-W AP/M/L	0.0050	860000	6200	2000
HACK & SQUIRT	---	---	---	---
INJECTION BAR	---	---	---	---
<u>Routine-Worst Case Exposures</u>				
PILOT	0.3507	12000	89	29
MIXER/LOADER	0.7778	5600	41	13
SUPERVISOR	0.0608	71000	510	160
OBSERVER	0.0108	400000	2900	920
BACKPACK	0.9665	4500	32	10
R-O-W SPRAYER	0.0828	52000	370	120
R-O-W MIX/L	0.1115	39000	280	90
R-O-W AP/M/L	0.0930	46000	330	110
HACK & SQUIRT	---	---	---	---
INJECTION BAR	---	---	---	---

^aThe plus sign (+) means "greater than."

Table C-28

Margins of Safety for Workers Wearing
Protective Clothing and Using Hexazinone

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (1690.0)	SYSTEMIC NOEL (10.00)	REPRODUCTIVE NOEL (125.00)
<u>Routine-Realistic Exposures</u>				
PILOT	0.0211	80000	470	5900
MIXER/LOADER	0.0526	32000	190	2400
SUPERVISOR	0.0032	520000	3100	39000
OBSERVER	0.0007	1000000+	15000	180000
BACKPACK	0.0576	29000	170	2200
R-O-W SPRAYER	0.0027	630000	3700	47000
R-O-W MIX/L	0.0064	270000	1600	20000
R-O-W AP/M/L	0.0063	270000	1600	20000
HACK & SQUIRT	---	---	---	---
INJECTION BAR	---	---	---	---
<u>Routine-Worst Case Exposures</u>				
PILOT	0.2104	8000	48	590
MIXER/LOADER	0.4667	3600	21	270
SUPERVISOR	0.0365	46000	270	3400
OBSERVER	0.0065	260000	1500	19000
BACKPACK	0.5799	2900	17	220
R-O-W SPRAYER	0.0993	17000	100	1300
R-O-W MIX/L	0.1338	13000	75	930
R-O-W AP/M/L	0.1116	15000	90	1100
HACK & SQUIRT	---	---	---	---
INJECTION BAR	---	---	---	---

^aThe plus sign (+) means "greater than."

Table C-29

Margins of Safety for Workers Wearing
Protective Clothing and Using Picloram

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (8200.0)	SYSTEMIC NOEL (7.00)	REPRODUCTIVE NOEL (50.00)
<u>Routine-Realistic Exposures</u>				
PILOT	0.0004	1000000+	17000	120000
MIXER/LOADER	0.0010	1000000+	6900	49000
SUPERVISOR	0.0001	1000000+	110000	810000
OBSERVER	0.0000	1000000+	530000	1000000+
BACKPACK	0.0025	1000000+	2800	20000
R-O-W SPRAYER	0.0001	1000000+	140000	970000
R-O-W MIX/L	0.0001	1000000+	57000	410000
R-O-W AP/M/L	0.0001	1000000+	58000	420000
HACK & SQUIRT	0.0011	1000000+	6200	44000
INJECTION BAR	0.0005	1000000+	15000	110000
<u>Routine-Worst Case Exposures</u>				
PILOT	0.0168	490000	420	3000
MIXER/LOADER	0.0373	220000	190	1300
SUPERVISOR	0.0029	1000000+	2400	17000
OBSERVER	0.0005	1000000+	13000	96000
BACKPACK	0.0371	220000	190	1300
R-O-W SPRAYER	0.0016	1000000+	4400	31000
R-O-W MIX/L	0.0021	1000000+	3300	23000
R-O-W AP/M/L	0.0018	1000000+	3900	28000
HACK & SQUIRT	0.0128	640000	550	3900
INJECTION BAR	0.0037	1000000+	1900	14000

^aThe plus sign (+) means "greater than."

Table C-30

Margins of Safety for Workers Wearing
Protective Clothing and Using Simazine

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (5000.0)	SYSTEMIC NOEL (5.00)	REPRODUCTIVE NOEL (5.00)
<u>Routine-Realistic Exposures</u>				
ILOT	0.0338	150000	150	150
MIXER/LOADER	0.0842	59000	59	59
SUPERVISOR	0.0052	970000	970	970
OBSERVER	0.0011	1000000+	4600	4600
BACKPACK	0.1029	49000	49	49
R-O-W SPRAYER	0.0021	1000000+	2300	2300
R-O-W MIX/L	0.0051	980000	980	980
R-O-W AP/M/L	0.0050	1000000	1000	1000
HACK & SQUIRT	---	---	---	---
INJECTION BAR	---	---	---	---
<u>Routine-Worst Case Exposures</u>				
PILOT	0.3507	14000	14	14
MIXER/LOADER	0.7778	6400	6.4	6.4
SUPERVISOR	0.0608	82000	82	82
OBSERVER	0.0108	460000	460	460
BACKPACK	0.8892	5600	5.6	5.6
R-O-W SPRAYER	0.0761	66000	66	66
R-O-W MIX/L	0.1026	49000	49	49
R-O-W AP/M/L	0.0856	58000	58	58
HACK & SQUIRT	---	---	---	---
INJECTION BAR	---	---	---	---

^aThe plus sign (+) means "greater than."

Table C-31

Margins of Safety for Workers Wearing
Protective Clothing and Using Tebuthiuron

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (644.0)	SYSTEMIC NOEL (12.50)	REPRODUCTIVE NOEL (90.00)
<u>Routine-Realistic Exposures</u>				
PILOT	0.0084	76000	1500	11000
MIXER/LOADER	0.0211	31000	590	4300
SUPERVISOR	0.0013	500000	9700	70000
OBSERVER	0.0003	1000000+	46000	330000
BACKPACK	0.0772	8300	160	1200
R-O-W SPRAYER	0.0024	270000	5300	38000
R-O-W MIX/L	0.0056	110000	2200	16000
R-O-W AP/M/L	0.0055	120000	2300	16000
HACK & SQUIRT	---	---	---	---
INJECTION BAR	---	---	---	---
<u>Routine-Worst Case Exposures</u>				
PILOT	0.4208	1500	30	210
MIXER/LOADER	0.9334	690	13	96
SUPERVISOR	0.0729	8800	170	1200
OBSERVER	0.0130	50000	960	6900
BACKPACK	1.1598	560	11	78
R-O-W SPRAYER	0.0761	8500	160	1200
R-O-W MIX/L	0.1026	6300	120	880
R-O-W AP/M/L	0.0856	7500	150	1100
HACK & SQUIRT	---	---	---	---
INJECTION BAR	---	---	---	---

^aThe plus sign (+) means "greater than."

Table C-32

Margins of Safety for Workers Wearing
Protective Clothing and Using Triclopyr

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (630.0)	SYSTEMIC NOEL (2.50)	REPRODUCTIVE NOEL (10.00)
<u>Routine-Realistic Exposures</u>				
PILOT	0.0169	37000	150	590
MIXER/LOADER	0.0421	15000	59	240
SUPERVISOR	0.0026	240000	970	3900
OBSERVER	0.0005	1000000+	4600	18000
BACKPACK	0.1029	6100	24	97
R-O-W SPRAYER	0.0021	290000	1200	4700
R-O-W MIX/L	0.0051	120000	490	2000
R-O-W AP/M/L	0.0050	130000	500	2000
HACK & SQUIRT	0.0472	13000	53	210
INJECTION BAR	0.0192	33000	130	520
<u>Routine-Worst Case Exposures</u>				
PILOT	0.5611	1100	4.5	18
MIXER/LOADER	1.2445	510	2.0	8.0
SUPERVISOR	0.0972	6500	26	100
OBSERVER	0.0173	36000	140	580
BACKPACK	1.5464	410	1.6	6.5
R-O-W SPRAYER	0.1324	4800	19	76
R-O-W MIX/L	0.1784	3500	14	56
R-O-W AP/M/L	0.1488	4200	17	67
HACK & SQUIRT	0.5350	1200	4.7	19
INJECTION BAR	0.1528	4100	16	65

^aThe plus sign (+) means "greater than."

Table C-33

Margins of Safety for Exposed Members of the Public
 Aerial Routine-Realistic Scenario
 Amitrole Sprayed on 40 acres by Helicopter

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE	LD50	SYSTEMIC	REPRODUCTIVE
	(MG/KG/DAY)	(4080.0)	NOEL (0.03)	NOEL (5.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0000	1000000+	160000	1000000+
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0000	1000000+	770	150000
DRINKING WATER	0.0019	1000000+	13	2700
EATING BERRIES	0.0011	1000000+	23	4600
EATING VEGETS.	0.0022	1000000+	12	2300
EATING DEER	0.0001	1000000+	170	34000
EATING BIRD	0.0005	1000000+	51	10000
EATING FISH	0.0008	1000000+	33	6600
<u>For Combined Routes of Exposure</u>				
HIKER	0.0019	1000000+	13	2700
BERRY PICKER	0.0030	1000000+	8.3	1700
HUNTER	0.0025	1000000+	9.9	2000
FISHERMAN	0.0026	1000000+	9.5	1900
RESIDENT	0.0040	1000000	6.2	1200

^aThe plus sign (+) means "greater than."

Table C-34

Margins of Safety for Exposed Members of the Public
Backpack Routine-Realistic Scenario
Amitrole Sprayed on 6 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (4080.0)	SYSTEMIC NOEL (0.03)	REPRODUCTIVE NOEL (5.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0000	1000000+	14000	1000000+
VEGETATION CONTACT				
HIKER	0.0000	1000000+	980000	1000000+
PICKER	0.0000	1000000+	3500	710000
DRINKING WATER	0.0002	1000000+	150	30000
EATING BERRIES	0.0003	1000000+	82	16000
EATING VEGETS.	0.0006	1000000+	41	8200
EATING DEER	0.0000	1000000+	660	130000
EATING BIRD	0.0001	1000000+	230	46000
EATING FISH	0.0001	1000000+	380	75000
<u>For Combined Routes of Exposure:</u>				
HIKER	0.0002	1000000+	150	30000
BERRY PICKER	0.0005	1000000+	52	10000
HUNTER	0.0003	1000000+	79	16000
FISHERMAN	0.0002	1000000+	110	21000
RESIDENT	0.0008	1000000+	32	6400

^aThe plus sign (+) means "greater than."

Table C-35

Margins of Safety for Exposed Members of the Public
 Right-of-Way Routine-Realistic Scenario
 Amitrole Sprayed on 12 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (4080.0)	SYSTEMIC NOEL (0.03)	REPRODUCTIVE NOEL (5.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0000	1000000+	72000	1000000+
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0000	1000000+	13000	1000000+
DRINKING WATER	0.0001	1000000+	340	67000
EATING BERRIES	0.0001	1000000+	240	49000
EATING VEGETS.	0.0002	1000000+	120	24000
EATING DEER	0.0000	1000000+	2000	410000
EATING BIRD	0.0000	1000000+	820	160000
EATING FISH	0.0000	1000000+	840	170000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0001	1000000+	330	67000
BERRY PICKER	0.0002	1000000+	140	28000
HUNTER	0.0001	1000000+	210	43000
FISHERMAN	0.0001	1000000+	240	48000
RESIDENT	0.0003	1000000+	90	18000

^aThe plus sign (+) means "greater than."

Table C-36

Margins of Safety for Exposed Members of the Public
 Aerial Routine-Worst Case Scenario
 Amitrole Sprayed on 400 acres by Fixed Wing

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (4080.0)	SYSTEMIC NOEL (0.03)	REPRODUCTIVE NOEL (5.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0002	1000000+	150	29000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	10000	1000000+
PICKER	0.0004	1000000+	57	11000
DRINKING WATER	0.0127	320000	2.0	390
EATING BERRIES	0.0104	390000	2.4	480
EATING VEGETS.	0.0208	200000	1.2	240
EATING DEER	0.0015	1000000+	16	3300
EATING BIRD	0.0063	650000	4.0	800
EATING FISH	0.0051	800000	4.9	990
<u>For Combined Routes of Exposure</u>				
HIKER	0.0129	320000	1.9	390
BERRY PICKER	0.0237	170000	1.1	210
HUNTER	0.0206	200000	1.2	240
FISHERMAN	0.0179	230000	1.4	280
RESIDENT	0.0337	120000	-1.3	150

^aThe plus sign (+) means "greater than."

Table C-37

Margins of Safety for Exposed Members of the Public
 Backpack Routine-Worst Case Scenario
 Amitrole Sprayed on 60 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (4080.0)	SYSTEMIC NOEL (0.03)	REPRODUCTIVE NOEL (5.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0000	1000000+	2900	580000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	200000	1000000+
PICKER	0.0000	1000000+	1100	230000
DRINKING WATER	0.0005	1000000+	52	10000
EATING BERRIES	0.0009	1000000+	27	5400
EATING VEGETS.	0.0018	1000000+	14	2700
EATING DEER	0.0001	1000000+	220	43000
EATING BIRD	0.0003	1000000+	74	15000
EATING FISH	0.0002	1000000+	130	26000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0005	1000000+	51	10000
BERRY PICKER	0.0014	1000000+	17	3500
HUNTER	0.0009	1000000+	27	5300
FISHERMAN	0.0007	1000000+	37	7400
RESIDENT	0.0023	1000000+	11	2100

^aThe plus sign (+) means "greater than."

Table C-38

Margins of Safety for Exposed Members of the Public
 Right of Way Routine-Worst Case Scenario
 Amitrole Sprayed on 40 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (4080.0)	SYSTEMIC NOEL (0.03)	REPRODUCTIVE NOEL (5.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0000	1000000+	5200	1000000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	360000	1000000+
PICKER	0.0000	1000000+	2000	400000
DRINKING WATER	0.0004	1000000+	63	13000
EATING BERRIES	0.0006	1000000+	40	8100
EATING VEGETS.	0.0012	1000000+	20	4000
EATING DEER	0.0001	1000000+	330	66000
EATING BIRD	0.0002	1000000+	130	25000
EATING FISH	0.0002	1000000+	160	31000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0004	1000000+	62	12000
BERRY PICKER	0.0010	1000000+	24	4800
HUNTER	0.0007	1000000+	37	7400
FISHERMAN	0.0006	1000000+	44	8900
RESIDENT	0.0016	1000000+	15	3000

^aThe plus sign (+) means "greater than."

Table C-39

Margins of Safety for Exposed Member of the Public
 Aerial Routine-Realistic Scenario
 Asulam Sprayed on 40 acres by Helicopter

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (4000.0)	SYSTEMIC NOEL (50.00)	REPRODUCTIVE NOEL (50.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0000	1000000+	1000000+	1000000+
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0039	1000000	13000	13000
DRINKING WATER	0.0023	1000000+	22000	22000
EATING BERRIES	0.0013	1000000+	39000	39000
EATING VEGETS.	0.0026	1000000+	19000	19000
EATING DEER	0.0002	1000000+	270000	270000
EATING BIRD	0.0007	1000000+	76000	76000
EATING FISH	0.0009	1000000+	55000	55000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0023	1000000+	22000	22000
BERRY PICKER	0.0075	540000	6700	6700
HUNTER	0.0031	1000000+	16000	16000
FISHERMAN	0.0032	1000000+	16000	16000
RESIDENT	0.0049	820000	10000	10000

^aThe plus sign (+) means "greater than."

Table C-40

Margins of Safety for Exposed Members of the Public
 Backpack Routine-Realistic Scenario
 Asulam Sprayed on 6 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (4000.0)	SYSTEMIC NOEL (50.00)	REPRODUCTIVE NOEL (50.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0001	1000000+	470000	470000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0004	1000000+	120000	120000
DRINKING WATER	0.0001	1000000+	500000	500000
EATING BERRIES	0.0002	1000000+	270000	270000
EATING VEGETS.	0.0004	1000000+	140000	140000
EATING DEER	0.0000	1000000+	1000000+	1000000+
EATING BIRD	0.0001	1000000+	680000	680000
EATING FISH	0.0000	1000000+	1000000+	1000000+
<u>For Combined Routes of Exposure</u>				
HIKER	0.0002	1000000+	240000	240000
BERRY PICKER	0.0008	1000000+	62000	62000
HUNTER	0.0003	1000000+	160000	160000
FISHERMAN	0.0002	1000000+	200000	200000
RESIDENT	0.0006	1000000+	87000	87000

^aThe plus sign (+) means "greater than."

Table C-41

Margins of Safety for Exposed Members of the Public
 Right-of-Way Routine-Realistic Scenario
 Asulam Sprayed on 12 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (4000.0)	SYSTEMIC NOEL (50.00)	REPRODUCTIVE NOEL (50.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0000	1000000+	1000000+	1000000+
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0002	1000000+	220000	220000
DRINKING WATER	0.0001	1000000+	560000	560000
EATING BERRIES	0.0001	1000000+	410000	410000
EATING VEGETS.	0.0002	1000000+	200000	200000
EATING DEER	0.0000	1000000+	1000000+	1000000+
EATING BIRD	0.0000	1000000+	1000000+	1000000+
EATING FISH	0.0000	1000000+	1000000+	1000000+
<u>For Combined Routes of Exposure</u>				
HIKER	0.0001	1000000+	380000	380000
BERRY PICKER	0.0005	1000000+	100000	100000
HUNTER	0.0002	1000000+	270000	270000
FISHERMAN	0.0002	1000000+	300000	300000
RESIDENT	0.0004	1000000+	130000	130000

^aThe plus sign (+) means "greater than."

Table C-42

Margins of Safety for Exposed Members of the Public
Aerial Routine-Worst Case Scenario
Asulam Sprayed on 400 acres by Fixed Wing

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (4000.0)	SYSTEMIC NOEL (50.00)	REPRODUCTIVE NOEL (50.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0142	280000	3500	3500
VEGETATION CONTACT				
HIKER	0.0002	1000000+	250000	250000
PICKER	0.0366	110000	1400	1400
DRINKING WATER	0.0106	380000	4700	4700
EATING BERRIES	0.0087	460000	5700	5700
EATING VEGETS.	0.0174	230000	2900	2900
EATING DEER	0.0014	1000000+	37000	37000
EATING BIRD	0.0059	670000	8400	8400
EATING FISH	0.0042	940000	12000	12000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0250	160000	2000	2000
BERRY PICKER	0.0700	57000	710	710
HUNTER	0.0323	120000	1500	1500
FISHERMAN	0.0292	140000	1700	1700
RESIDENT	0.0424	94000	1200	1200

^aThe plus sign (+) means "greater than."

Table C-43

Margins of Safety for Exposed Members of the Public
 Backpack Routine-Worst Case Scenario
 Asulam Sprayed on 60 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (4000.0)	SYSTEMIC NOEL (50.00)	REPRODUCTIVE NOEL (50.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0006	1000000+	87000	87000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0015	1000000+	34000	34000
DRINKING WATER	0.0003	1000000+	160000	160000
EATING BERRIES	0.0006	1000000+	81000	81000
EATING VEGETS.	0.0012	1000000+	41000	41000
EATING DEER	0.0001	1000000+	620000	620000
EATING BIRD	0.0003	1000000+	200000	200000
EATING FISH	0.0001	1000000+	390000	390000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0009	1000000+	56000	56000
BERRY PICKER	0.0030	1000000+	17000	17000
HUNTER	0.0012	1000000+	40000	40000
FISHERMAN	0.0010	1000000+	49000	49000
RESIDENT	0.0021	1000000+	23000	23000

^aThe plus sign (+) means "greater than."

Table C-44

Margins of Safety for Exposed Members of the Public
 Right of Way Routine-Worst Case Scenario
 Asulam Sprayed on 40 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (4000.0)	SYSTEMIC NOEL (50.00)	REPRODUCTIVE NOEL (50.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0003	1000000+	170000	170000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0008	1000000+	65000	65000
DRINKING WATER	0.0002	1000000+	200000	200000
EATING BERRIES	0.0004	1000000+	130000	130000
EATING VEGETS.	0.0008	1000000+	64000	64000
EATING DEER	0.0000	1000000+	1000000+	1000000+
EATING BIRD	0.0001	1000000+	360000	360000
EATING FISH	0.0001	1000000+	500000	500000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0006	1000000+	90000	90000
BERRY PICKER	0.0017	1000000+	29000	29000
HUNTER	0.0007	1000000+	68000	68000
FISHERMAN	0.0007	1000000+	77000	77000
RESIDENT	0.0013	1000000+	38000	38000

^aThe plus sign (+) means "greater than."

Table C-45

Margins of Safety for Exposed Members of the Public
 Aerial Routine-Realistic Scenario
 Atrazine Sprayed on 40 acres by Helicopter

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (1869.0)	SYSTEMIC NOEL (3.70)	REPRODUCTIVE NOEL (100.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0000	1000000+	120000	1000000+
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0061	310000	610	16000
DRINKING WATER	0.0035	530000	1000	28000
EATING BERRIES	0.0020	920000	1800	49000
EATING VEGETS.	0.0040	460000	910	25000
EATING DEER	0.0003	1000000+	13000	350000
EATING BIRD	0.0010	1000000+	3600	97000
EATING FISH	0.0071	260000	520	14000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0036	520000	1000	28000
BERRY PICKER	0.0117	160000	320	8600
HUNTER	0.0049	380000	760	20000
FISHERMAN	0.0106	180000	350	9400
RESIDENT	0.0076	250000	490	13000

^aThe plus sign (+) means "greater than."

Table C-46

Margins of Safety for Exposed Members of the Public
Backpack Routine-Realistic Scenario
Atrazine Sprayed on 6 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (1869.0)	SYSTEMIC NOEL (3.70)	REPRODUCTIVE NOEL (100.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0003	1000000+	14000	370000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	970000	1000000+
PICKER	0.0011	1000000+	3500	95000
DRINKING WATER	0.0002	1000000+	15000	400000
EATING BERRIES	0.0005	1000000+	8100	220000
EATING VEGETS.	0.0009	1000000+	4000	110000
EATING DEER	0.0001	1000000+	62000	1000000+
EATING BIRD	0.0002	1000000+	20000	540000
EATING FISH	0.0005	1000000+	7400	200000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0005	1000000+	7100	190000
BERRY PICKER	0.0020	920000	1800	49000
HUNTER	0.0008	1000000+	4800	130000
FISHERMAN	0.0010	1000000+	3600	98000
RESIDENT	0.0014	1000000+	2600	70000

^aThe plus sign (+) means "greater than."

Table C-47

Margins of Safety for Exposed Members of the Public
 Right-of-Way Routine-Realistic Scenario
 Atrazine Sprayed on 12 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (1869.0)	SYSTEMIC NOEL (3.70)	REPRODUCTIVE NOEL (100.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0001	1000000+	72000	1000000+
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0003	1000000+	13000	360000
DRINKING WATER	0.0001	1000000+	33000	900000
EATING BERRIES	0.0002	1000000+	24000	650000
EATING VEGETS.	0.0003	1000000+	12000	330000
EATING DEER	0.0000	1000000+	190000	1000000+
EATING BIRD	0.0001	1000000+	73000	1000000+
EATING FISH	0.0002	1000000+	17000	450000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0002	1000000+	23000	610000
BERRY PICKER	0.0006	1000000+	6200	170000
HUNTER	0.0002	1000000+	16000	430000
FISHERMAN	0.0004	1000000+	9600	260000
RESIDENT	0.0005	1000000+	7900	210000

^aThe plus sign (+) means "greater than."

Table C-48

Margins of Safety for Exposed Members of the Public
 Aerial Routine-Worst Case Scenario
 Atrazine Sprayed on 400 acres by Fixed Wing

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (1869.0)	SYSTEMIC NOEL (3.70)	REPRODUCTIVE NOEL (100.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0170	110000	220	5900
VEGETATION CONTACT				
HIKER	0.0002	1000000+	15000	410000
PICKER	0.0438	43000	85	2300
DRINKING WATER	0.0127	150000	290	7900
EATING BERRIES	0.0104	180000	350	9600
EATING VEGETS.	0.0208	90000	180	4800
EATING DEER	0.0016	1000000+	2300	61000
EATING BIRD	0.0071	260000	520	14000
EATING FISH	0.0254	74000	150	3900
<u>For Combined Routes of Exposure</u>				
HIKER	0.0299	62000	120	3300
BERRY PICKER	0.0839	22000	44	1200
HUNTER	0.0386	48000	96	2600
FISHERMAN	0.0553	34000	67	1800
RESIDENT	0.0508	37000	73	2000

^aThe plus sign (+) means "greater than."

Table C-49

Margins of Safety for Exposed Members of the Public
 Backpack Routine-Worst Case Scenario
 Atrazine Sprayed on 60 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (1869.0)	SYSTEMIC NOEL (3.70)	REPRODUCTIVE NOEL (100.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0007	1000000+	5400	150000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	380000	1000000+
PICKER	0.0018	1000000+	2100	57000
DRINKING WATER	0.0004	1000000+	9700	260000
EATING BERRIES	0.0007	1000000+	5000	140000
EATING VEGETS.	0.0015	1000000+	2500	68000
EATING DEER	0.0001	1000000+	38000	1000000
EATING BIRD	0.0003	1000000+	12000	330000
EATING FISH	0.0008	1000000+	4800	130000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0011	1000000+	3400	93000
BERRY PICKER	0.0036	520000	1000	28000
HUNTER	0.0015	1000000+	2500	68000
FISHERMAN	0.0018	1000000	2000	54000
RESIDENT	0.0026	730000	1400	39000

^aThe plus sign (+) means "greater than."

Table C-50

Margins of Safety for Exposed Members of the Public
 Right of Way Routine-Worst Case Scenario
 Atrazine Sprayed on 40 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (1869.0)	SYSTEMIC NOEL (3.70)	REPRODUCTIVE NOEL (100.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0005	1000000+	7300	200000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	510000	1000000+
PICKER	0.0013	1000000+	2800	76000
DRINKING WATER	0.0004	1000000+	8700	240000
EATING BERRIES	0.0007	1000000+	5600	150000
EATING VEGETS.	0.0013	1000000+	2800	76000
EATING DEER	0.0001	1000000+	44000	1000000+
EATING BIRD	0.0002	1000000+	16000	430000
EATING FISH	0.0008	1000000+	4400	120000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0009	1000000+	3900	110000
BERRY PICKER	0.0029	640000	1300	34000
HUNTER	0.0013	1000000+	2900	80000
FISHERMAN	0.0018	1000000	2100	56000
RESIDENT	0.0023	830000	1600	44000

^aThe plus sign (+) means "greater than."

Table C-51

Margins of Safety for Exposed Members of the Public
 Aerial Routine-Realistic Scenario
 Bromacil Sprayed on 40 acres by Helicopter

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (3998.0)	SYSTEMIC NOEL (6.25)	REPRODUCTIVE NOEL (7.92)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	----	----	----	----
VEGETATION CONTACT				
HIKER	----	----	----	----
PICKER	----	----	----	----
DRINKING WATER	----	----	----	----
EATING BERRIES	----	----	----	----
EATING VEGETS.	----	----	----	----
EATING DEER	----	----	----	----
EATING BIRD	----	----	----	----
EATING FISH	----	----	----	----
<u>For Combined Routes of Exposure</u>				
HIKER	----	----	----	----
BERRY PICKER	----	----	----	----
HUNTER	----	----	----	----
FISHERMAN	----	----	----	----
RESIDENT	----	----	----	----

Table C-52

Margins of Safety for Exposed Members of the Public
Backpack Routine-Realistic Scenario
Bromacil Sprayed on 6 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (3998.0)	SYSTEMIC NOEL (6.25)	REPRODUCTIVE NOEL (7.92)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0004	1000000+	18000	22000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0014	1000000+	4400	5600
DRINKING WATER	0.0003	1000000+	19000	24000
EATING BERRIES	0.0006	1000000+	10000	13000
EATING VEGETS.	0.0012	1000000+	5100	6500
EATING DEER	0.0001	1000000+	78000	99000
EATING BIRD	0.0002	1000000+	26000	32000
EATING FISH	0.0001	1000000+	47000	60000
<u>For Combined Routes of Exposure:</u>				
HIKER	0.0007	1000000+	9000	11000
BERRY PICKER	0.0027	1000000+	2300	2900
HUNTER	0.0010	1000000+	6100	7800
FISHERMAN	0.0008	1000000+	7600	9600
RESIDENT	0.0019	1000000+	3300	4100

^aThe plus sign (+) means "greater than."

Table C-53

Margins of Safety for Exposed Members of the Public
 Right-of-Way Routine-Realistic Scenario
 Bromacil Sprayed on 12 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (3998.0)	SYSTEMIC NOEL (6.25)	REPRODUCTIVE NOEL (7.92)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0001	1000000+	91000	110000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0004	1000000+	17000	21000
DRINKING WATER	0.0001	1000000+	42000	53000
EATING BERRIES	0.0002	1000000+	31000	39000
EATING VEGETS.	0.0004	1000000+	15000	19000
EATING DEER	0.0000	1000000+	250000	310000
EATING BIRD	0.0001	1000000+	92000	120000
EATING FISH	0.0001	1000000+	100000	130000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0002	1000000+	29000	36000
BERRY PICKER	0.0008	1000000+	7900	10000
HUNTER	0.0003	1000000+	20000	25000
FISHERMAN	0.0003	1000000+	22000	28000
RESIDENT	0.0006	1000000+	10000	13000

^aThe plus sign (+) means "greater than."

Table C-54

Margins of Safety for Exposed Members of the Public
 Aerial Routine-Worst Case Scenario
 Bromacil Sprayed on 400 acres by Fixed Wing

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (3998.0)	SYSTEMIC NOEL (6.25)	REPRODUCTIVE NOEL (7.92)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	---	---	---	---
VEGETATION CONTACT				
HIKER	---	---	---	---
PICKER	---	---	---	---
DRINKING WATER	---	---	---	---
EATING BERRIES	---	---	---	---
EATING VEGETS.	---	---	---	---
EATING DEER	---	---	---	---
EATING BIRD	---	---	---	---
EATING FISH	---	---	---	---
<u>For Combined Routes of Exposure</u>				
HIKER	---	---	---	---
BERRY PICKER	---	---	---	---
HUNTER	---	---	---	---
FISHERMAN	---	---	---	---
RESIDENT	---	---	---	---

Table C-55

Margins of Safety for Exposed Members of the Public
Backpack Routine-Worst Case Scenario
Bromacil Sprayed on 60 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (3998.0)	SYSTEMIC NOEL (6.25)	REPRODUCTIVE NOEL (7.92)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0017	1000000+	3600	4600
VEGETATION CONTACT				
HIKER	0.0000	1000000+	250000	320000
PICKER	0.0044	910000	1400	1800
DRINKING WATER	0.0010	1000000+	6500	8300
EATING BERRIES	0.0018	1000000+	3400	4300
EATING VEGETS.	0.0037	1000000+	1700	2100
EATING DEER	0.0002	1000000+	26000	33000
EATING BIRD	0.0008	1000000+	8200	10000
EATING FISH	0.0004	1000000+	16000	21000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0027	1000000+	2300	2900
BERRY PICKER	0.0089	450000	700	890
HUNTER	0.0037	1000000+	1700	2100
FISHERMAN	0.0031	1000000+	2000	2600
RESIDENT	0.0064	630000	980	1200

^aThe plus sign (+) means "greater than."

Table C-56

Margins of Safety for Exposed Members of the Public
 Right of Way Routine-Worst Case Scenario
 Bromacil Sprayed on 40 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (3998.0)	SYSTEMIC NOEL (6.25)	REPRODUCTIVE NOEL (7.92)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0006	1000000+	10000	13000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	730000	920000
PICKER	0.0015	1000000+	4000	5100
DRINKING WATER	0.0005	1000000+	13000	16000
EATING BERRIES	0.0008	1000000+	8100	10000
EATING VEGETS.	0.0016	1000000+	4000	5100
EATING DEER	0.0001	1000000+	64000	81000
EATING BIRD	0.0003	1000000+	23000	29000
EATING FISH	0.0002	1000000+	31000	40000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0011	1000000+	5700	7200
BERRY PICKER	0.0034	1000000+	1800	2300
HUNTER	0.0015	1000000+	4200	5400
FISHERMAN	0.0013	1000000+	4800	6100
RESIDENT	0.0027	1000000+	2400	3000

^aThe plus sign (+) means "greater than."

Table C-57

Margins of Safety for Exposed Members of the Public
 Aerial Routine-Realistic Scenario
 2,4-D Sprayed on 40 acres by Helicopter

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (375.0)	SYSTEMIC NOEL (1.00)	REPRODUCTIVE NOEL (5.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0000	1000000+	83000	1000000+
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0024	150000	410	2000
DRINKING WATER	0.0024	160000	420	2200
EATING BERRIES	0.0013	280000	740	3800
EATING VEGETS.	0.0027	140000	370	1900
EATING DEER	0.0002	1000000+	5300	26000
EATING BIRD	0.0007	570000	1500	7600
EATING FISH	0.0009	400000	1100	5400
<u>For Combined Routes of Exposure:</u>				
HIKER	0.0024	160000	420	2200
BERRY PICKER	0.0061	61000	160	820
HUNTER	0.0032	120000	310	1600
FISHERMAN	0.0033	110000	300	1500
RESIDENT	0.0051	74000	200	980

^aThe plus sign (+) means "greater than."

Table C-58

Margins of Safety for Exposed Members of the Public
 Backpack Routine-Realistic Scenario
 2,4-D Sprayed on 6 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (375.0)	SYSTEMIC NOEL (1.00)	REPRODUCTIVE NOEL (5.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0001	1000000+	9400	4600
VEGETATION CONTACT				
HIKER	0.0000	1000000+	650000	1000000+
PICKER	0.0004	890000	2400	12000
DRINKING WATER	0.0002	1000000+	6000	30000
EATING BERRIES	0.0003	1000000+	3300	16000
EATING VEGETS.	0.0006	610000	1600	8200
EATING DEER	0.0000	1000000+	26000	130000
EATING BIRD	0.0001	1000000+	8500	42000
EATING FISH	0.0001	1000000+	15000	76000
<u>For Combined Routes of Exposure:</u>				
HIKER	0.0003	1000000+	3600	18000
BERRY PICKER	0.0010	370000	1000	5000
HUNTER	0.0004	870000	2300	12000
FISHERMAN	0.0003	1000000+	2900	15000
RESIDENT	0.0009	420000	1100	5600

Table C-59

Margins of Safety for Exposed Members of the Public
 Right-of-Way Routine-Realistic Scenario
 2,4-D Sprayed on 12 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (375.0)	SYSTEMIC NOEL (1.00)	REPRODUCTIVE NOEL (5.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0000	1000000+	39000	19000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0001	1000000+	7100	36000
DRINKING WATER	0.0001	1000000+	11000	54000
EATING BERRIES	0.0001	1000000+	7800	40000
EATING VEGETS.	0.0003	1000000+	3900	20000
EATING DEER	0.0000	1000000+	64000	1000000+
EATING BIRD	0.0000	1000000+	25000	120000
EATING FISH	0.0000	1000000+	27000	130000
<u>For Combined Routes of Exposure:</u>				
HIKER	0.0001	1000000+	8400	42000
BERRY PICKER	0.0004	970000	2600	13000
HUNTER	0.0002	1000000+	5700	28000
FISHERMAN	0.0002	1000000+	6400	32000
RESIDENT	0.0004	1000000	2700	13000

^aThe plus sign (+) means "greater than."

Table C-60

Margins of Safety for Exposed Members of the Public
 Aerial Routine-Worst Case Scenario
 2,4-D Sprayed on 400 acres by Fixed Wing

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE	LD50	SYSTEMIC	REPRODUCTIVE
	(MG/KG/DAY)	(375.0)	NOEL (1.00)	NOEL (5.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0102	37000	98	500
VEGETATION CONTACT				
HIKER	0.0001	1000000+	6800	34000
PICKER	0.0263	14000	38	190
DRINKING WATER	0.0127	30000	79	400
EATING BERRIES	0.0104	36000	96	480
EATING VEGETS.	0.0208	18000	48	240
EATING DEER	0.0016	240000	630	3200
EATING BIRD	0.0068	55000	150	740
EATING FISH	0.0051	74000	200	980
<u>For Combined Routes of Exposure</u>				
HIKER	0.0230	16000	43	220
BERRY PICKER	0.0596	6300	17	84
HUNTER	0.0314	12000	32	160
FISHERMAN	0.0281	13000	36	180
RESIDENT	0.0439	8500	23	110

^aThe plus sign (+) means "greater than."

Table C-61

Margins of Safety for Exposed Members of the Public
 Backpack Routine-Worst Case Scenario
 2,4-D Sprayed on 60 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (375.0)	SYSTEMIC NOEL (1.00)	REPRODUCTIVE NOEL (5.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0004	910000	2400	12000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	170000	1000000+
PICKER	0.0011	350000	940	4800
DRINKING WATER	0.0004	980000	2600	13000
EATING BERRIES	0.0007	510000	1400	68000
EATING VEGETS.	0.0015	250000	680	34000
EATING DEER	0.0001	1000000+	11000	52000
EATING BIRD	0.0003	1000000+	3400	17000
EATING FISH	0.0002	1000000+	6500	32000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0008	470000	1300	6200
BERRY PICKER	0.0026	140000	390	1900
HUNTER	0.0012	320000	840	4200
FISHERMAN	0.0010	390000	1100	5200
RESIDENT	0.0023	160000	440	2200

^aThe plus sign (+) means "greater than."

Table C-62

Margins of Safety for Exposed Members of the Public
 Right of Way Routine-Worst Case Scenario
 2,4-D Sprayed on 40 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (375.0)	SYSTEMIC NOEL (1.00)	REPRODUCTIVE NOEL (5.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0001	1000000+	6800	34000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	470000	1000000+
PICKER	0.0004	990000	2600	13000
DRINKING WATER	0.0002	1000000+	4900	24000
EATING BERRIES	0.0003	1000000+	3100	16000
EATING VEGETS.	0.0006	590000	1600	7800
EATING DEER	0.0000	1000000+	25000	130000
EATING BIRD	0.0001	1000000+	9300	46000
EATING FISH	0.0001	1000000+	12000	62000
<u>For Combined Routes of Exposure:</u>				
HIKER	0.0004	1000000+	2800	14000
BERRY PICKER	0.0010	360000	950	4800
HUNTER	0.0005	750000	2000	10000
FISHERMAN	0.0004	860000	2300	11000
RESIDENT	0.0010	380000	1000	5000

^aThe plus sign (+) means "greater than."

Table C-63

Margins of Safety for Exposed Members of the Public
 Aerial Routine-Realistic Scenario
 2,4-DP Sprayed on 40 acres by Helicopter

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (532.0)	SYSTEMIC NOEL (5.00)	REPRODUCTIVE NOEL (6.25)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0000	1000000+	490000	610000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0021	260000	2400	3000
DRINKING WATER	0.0019	280000	2700	3300
EATING BERRIES	0.0011	490000	4600	5800
EATING VEGETS.	0.0022	250000	2300	2900
EATING DEER	0.0002	1000000+	33000	41000
EATING BIRD	0.0005	1000000	9500	12000
EATING FISH	0.0008	710000	6600	8300
<u>For Combined Routes of Exposure</u>				
HIKER	0.0019	280000	2600	3300
BERRY PICKER	0.0050	110000	990	1200
HUNTER	0.0026	210000	1900	2400
FISHERMAN	0.0026	200000	1900	2400
RESIDENT	0.0041	130000	1200	1500

^aThe plus sign (+) means "greater than."

Table C-64

Margins of Safety for Exposed Members of the Public
 Backpack Routine-Realistic Scenario
 2,4-DP Sprayed on 6 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (532.0)	SYSTEMIC NOEL (5.00)	REPRODUCTIVE NOEL (6.25)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0001	1000000+	44000	55000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0005	1000000+	11000	14000
DRINKING WATER	0.0002	1000000+	30000	38000
EATING BERRIES	0.0003	1000000+	16000	20000
EATING VEGETS.	0.0006	870000	8200	10000
EATING DEER	0.0000	1000000+	130000	160000
EATING BIRD	0.0001	1000000+	43000	53000
EATING FISH	0.0001	1000000+	75000	94000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0003	1000000+	18000	22000
BERRY PICKER	0.0010	510000	4800	6000
HUNTER	0.0004	1000000+	11000	14000
FISHERMAN	0.0003	1000000+	14000	18000
RESIDENT	0.0009	600000	5600	7000

^aThe plus sign (+) means "greater than."

Table C-65

Margins of Safety for Exposed Members of the Public
 Right-of-Way Routine-Realistic Scenario
 2,4-DP Sprayed on 12 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (532.0)	SYSTEMIC NOEL (5.00)	REPRODUCTIVE NOEL (6.25)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0000	1000000+	180000	230000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0001	1000000+	33000	42000
DRINKING WATER	0.0001	1000000+	54000	67000
EATING BERRIES	0.0001	1000000+	39000	49000
EATING VEGETS.	0.0003	1000000+	20000	24000
EATING DEER	0.0000	1000000+	320000	400000
EATING BIRD	0.0000	1000000+	120000	150000
EATING FISH	0.0000	1000000+	130000	170000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0001	1000000+	41000	52000
BERRY PICKER	0.0004	1000000+	13000	16000
HUNTER	0.0002	1000000+	28000	35000
FISHERMAN	0.0002	1000000+	32000	40000
RESIDENT	0.0004	1000000+	13000	17000

^aThe plus sign (+) means "greater than."

Table C-66

Margins of Safety for Exposed Members of the Public
 Aerial Routine-Worst Case Scenario
 2,4-DP Sprayed on 400 acres by Fixed Wing

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (532.0)	SYSTEMIC NOEL (5.00)	REPRODUCTIVE NOEL (6.25)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0068	78000	740	920
VEGETATION CONTACT				
HIKER	0.0001	1000000+	51000	64000
PICKER	0.0175	30000	290	360
DRINKING WATER	0.0079	67000	630	790
EATING BERRIES	0.0065	82000	770	960
EATING VEGETS.	0.0130	41000	380	480
EATING DEER	0.0010	530000	5000	6300
EATING BIRD	0.0043	130000	1200	1500
EATING FISH	0.0032	170000	1600	2000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0148	36000	340	420
BERRY PICKER	0.0387	14000	130	160
HUNTER	0.0201	27000	250	310
FISHERMAN	0.0180	30000	280	350
RESIDENT	0.0278	19000	180	220

^aThe plus sign (+) means "greater than."

Table C-67

Margins of Safety for Exposed Members of the Public
 Backpack Routine-Worst Case Scenario
 2,4-DP Sprayed on 60 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (532.0)	SYSTEMIC NOEL (5.00)	REPRODUCTIVE NOEL (6.25)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0005	1000000+	11000	13000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	740000	920000
PICKER	0.0012	440000	4100	5100
DRINKING WATER	0.0004	1000000+	12000	15000
EATING BERRIES	0.0008	670000	6300	7900
EATING VEGETS.	0.0016	340000	3200	3900
EATING DEER	0.0001	1000000+	49000	61000
EATING BIRD	0.0003	1000000+	16000	20000
EATING FISH	0.0002	1000000+	30000	38000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0009	600000	5600	7000
BERRY PICKER	0.0029	180000	1700	2200
HUNTER	0.0013	410000	3800	4800
FISHERMAN	0.0011	510000	4700	5900
RESIDENT	0.0025	220000	2000	2500

^aThe plus sign (+) means "greater than."

Table C-68
 Margins of Safety for Exposed Members of the Public
 Right of Way Routine-Worst Case Scenario
 2,4-DP Sprayed on 40 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE	LD50	SYSTEMIC	REPRODUCTIVE
	(MG/KG/DAY)	(532.0)	NOEL	NOEL
			(5.00)	(6.25)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0002	1000000+	26000	33000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0005	1000000+	10000	13000
DRINKING WATER	0.0002	1000000+	20000	25000
EATING BERRIES	0.0004	1000000+	13000	16000
EATING VEGETS.	0.0008	690000	6400	8100
EATING DEER	0.0000	1000000+	100000	130000
EATING BIRD	0.0001	1000000+	38000	47000
EATING FISH	0.0001	1000000+	50000	63000
<u>For Combined Routes of Exposure:</u>				
HIKER	0.0004	1000000+	11000	14000
BERRY PICKER	0.0013	400000	3800	4700
HUNTER	0.0006	850000	8000	10000
FISHERMAN	0.0005	980000	9200	12000
RESIDENT	0.0012	440000	4100	5100

^aThe plus sign (+) means "greater than."

Table C-69

Margins of Safety for Exposed Members of the Public
Aerial Routine-Realistic Scenario
Dalapon Sprayed on 40 acres by Helicopter

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (7577.0)	SYSTEMIC NOEL (15.00)	REPRODUCTIVE NOEL (300.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0000	1000000+	470000	1000000+
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0065	1000000+	2300	46000
DRINKING WATER	0.0038	1000000+	4000	80000
EATING BERRIES	0.0022	1000000+	7000	140000
EATING VEGETS.	0.0043	1000000+	3500	70000
EATING DEER	0.0003	1000000+	49000	970000
EATING BIRD	0.0011	1000000+	14000	270000
EATING FISH	0.0015	1000000+	10000	200000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0038	1000000+	3900	79000
BERRY PICKER	0.0124	610000	1200	24000
HUNTER	0.0052	1000000+	2900	58000
FISHERMAN	0.0053	1000000+	2800	57000
RESIDENT	0.0081	930000	1800	37000

^aThe plus sign (+) means "greater than."

Table C-70

Margins of Safety for Exposed Members of the Public
 Backpack Routine-Realistic Scenario
 Dalapon Sprayed on 6 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (7577.0)	SYSTEMIC NOEL (15.00)	REPRODUCTIVE NOEL (300.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0004	1000000+	42000	840000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0014	1000000+	11000	210000
DRINKING WATER	0.0003	1000000+	45000	900000
EATING BERRIES	0.0006	1000000+	25000	490000
EATING VEGETS.	0.0012	1000000+	12000	250000
EATING DEER	0.0001	1000000+	190000	1000000+
EATING BIRD	0.0002	1000000+	61000	1000000+
EATING FISH	0.0001	1000000+	110000	1000000+
<u>For Combined Routes of Exposure</u>				
HIKER	0.0007	1000000+	22000	430000
BERRY PICKER	0.0027	1000000+	5500	110000
HUNTER	0.0010	1000000+	15000	290000
FISHERMAN	0.0008	1000000+	18000	360000
RESIDENT	0.0019	1000000+	7800	160000

^aThe plus sign (+) means "greater than."

Table C-71

Margins of Safety for Exposed Members of the Public
 Right-of-Way Routine-Realistic Scenario
 Dalapon Sprayed on 12 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (7577.0)	SYSTEMIC NOEL (15.00)	REPRODUCTIVE NOEL (300.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0001	1000000+	220000	1000000+
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0004	1000000+	40000	800000
DRINKING WATER	0.0001	1000000+	100000	1000000+
EATING BERRIES	0.0002	1000000+	73000	1000000+
EATING VEGETS.	0.0004	1000000+	37000	730000
EATING DEER	0.0000	1000000+	590000	1000000+
EATING BIRD	0.0001	1000000+	220000	1000000+
EATING FISH	0.0001	1000000+	250000	1000000+
<u>For Combined Routes of Exposure</u>				
HIKER	0.0002	1000000+	69000	1000000+
BERRY PICKER	0.0008	1000000+	19000	380000
HUNTER	0.0003	1000000+	48000	960000
FISHERMAN	0.0003	1000000+	54000	1000000+
RESIDENT	0.0006	1000000+	24000	480000

^aThe plus sign (+) means "greater than."

Table C-72

Margins of Safety for Exposed Members of the Public
 Aerial Routine-Worst Case Scenario
 Dalapon Sprayed on 400 acres by Fixed Wing

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (7577.0)	SYSTEMIC NOEL (15.00)	REPRODUCTIVE NOEL (300.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0425	180000	350	7100
VEGETATION CONTACT				
HIKER	0.0006	1000000+	25000	490000
PICKER	0.1094	69000	140	2700
DRINKING WATER	0.0317	240000	470	9500
EATING BERRIES	0.0261	290000	580	12000
EATING VEGETS.	0.0521	150000	290	5800
EATING DEER	0.0041	1000000+	3700	74000
EATING BIRD	0.0178	430000	840	17000
EATING FISH	0.0127	600000	1200	24000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0748	100000	200	4000
BERRY PICKER	0.2097	36000	72	1400
HUNTER	0.0966	78000	160	3100
FISHERMAN	0.0874	87000	170	3400
RESIDENT	0.1269	60000	120	2400

^aThe plus sign (+) means "greater than."

Table C-73

Margins of Safety for Exposed Members of the Public
 Backpack Routine-Worst Case Scenario
 Dalapon Sprayed on 60 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (7577.0)	SYSTEMIC NOEL (15.00)	REPRODUCTIVE NOEL (300.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0021	1000000+	7300	150000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	510000	1000000+
PICKER	0.0053	1000000+	2800	57000
DRINKING WATER	0.0011	1000000+	13000	260000
EATING BERRIES	0.0022	1000000+	6800	140000
EATING VEGETS.	0.0044	1000000+	3400	68000
EATING DEER	0.0003	1000000+	52000	1000000
EATING BIRD	0.0009	1000000+	16000	330000
EATING FISH	0.0005	1000000+	33000	650000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0032	1000000+	4600	93000
BERRY PICKER	0.0107	710000	1400	28000
HUNTER	0.0044	1000000+	3400	68000
FISHERMAN	0.0037	1000000+	4100	81000
RESIDENT	0.0077	990000	2000	39000

^aThe plus sign (+) means "greater than."

Table C-74

Margins of Safety for Exposed Members of the Public
 Right of Way Routine-Worst Case Scenario
 Dalapon Sprayed on 40 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (7577.0)	SYSTEMIC NOEL (15.00)	REPRODUCTIVE NOEL (300.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0006	1000000+	25000	500000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0015	1000000+	9700	190000
DRINKING WATER	0.0005	1000000+	30000	600000
EATING BERRIES	0.0008	1000000+	19000	390000
EATING VEGETS.	0.0016	1000000+	9700	190000
EATING DEER	0.0001	1000000+	150000	1000000+
EATING BIRD	0.0003	1000000+	54000	1000000+
EATING FISH	0.0002	1000000+	75000	1000000+
<u>For Combined Routes of Exposure</u>				
HIKER	0.0011	1000000+	14000	270000
BERRY PICKER	0.0034	1000000+	4400	88000
HUNTER	0.0015	1000000+	10000	200000
FISHERMAN	0.0013	1000000+	11000	230000
RESIDENT	0.0027	1000000+	5600	110000

^aThe plus sign (+) means "greater than."

Table C-75

Margins of Safety for Exposed Members of the Public
 Aerial Routine-Realistic Scenario
 Dicamba Sprayed on 40 acres by Helicopter

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (757.0)	SYSTEMIC NOEL (25.00)	REPRODUCTIVE NOEL (2.50)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0000	1000000+	1000000+	620000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0008	930000	31000	3100
DRINKING WATER	0.0009	800000	27000	2700
EATING BERRIES	0.0005	1000000+	46000	4600
EATING VEGETS.	0.0011	700000	23000	2300
EATING DEER	0.0001	1000000+	330000	33000
EATING BIRD	0.0003	1000000+	96000	9600
EATING FISH	0.0004	1000000+	66000	6600
<u>For Combined Routes of Exposure</u>				
HIKER	0.0009	800000	26000	2600
BERRY PICKER	0.0023	330000	11000	1100
HUNTER	0.0013	590000	20000	2000
FISHERMAN	0.0013	570000	19000	1900
RESIDENT	0.0020	370000	12000	1200

^aThe plus sign (+) means "greater than."

Table C-76

Margins of Safety for Exposed Members of the Public
 Backpack Routine-Realistic Scenario
 Dicamba Sprayed on 6 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (757.0)	SYSTEMIC NOEL (25.00)	REPRODUCTIVE NOEL (2.50)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0000	1000000+	1000000+	110000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0001	1000000+	280000	28000
DRINKING WATER	0.0000	1000000+	600000	60000
EATING BERRIES	0.0001	1000000+	330000	33000
EATING VEGETS.	0.0002	1000000+	160000	16000
EATING DEER	0.0000	1000000+	1000000+	260000
EATING BIRD	0.0000	1000000+	860000	86000
EATING FISH	0.0000	1000000+	1000000+	150000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0001	1000000+	390000	39000
BERRY PICKER	0.0002	1000000+	110000	11000
HUNTER	0.0001	1000000+	240000	24000
FISHERMAN	0.0001	1000000+	310000	31000
RESIDENT	0.0002	1000000+	120000	12000

^aThe plus sign (+) means "greater than."

Table C-77

Margins of Safety for Exposed Members of the Public
 Right-of-Way Routine-Realistic Scenario
 Dicamba Sprayed on 12 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (757.0)	SYSTEMIC NOEL (25.00)	REPRODUCTIVE NOEL (2.50)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0000	1000000+	1000000+	290000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0000	1000000+	540000	54000
DRINKING WATER	0.0000	1000000+	670000	67000
EATING BERRIES	0.0001	1000000+	490000	49000
EATING VEGETS.	0.0001	1000000+	240000	24000
EATING DEER	0.0000	1000000+	1000000+	400000
EATING BIRD	0.0000	1000000+	1000000+	160000
EATING FISH	0.0000	1000000+	1000000+	170000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0000	1000000+	540000	54000
BERRY PICKER	0.0001	1000000+	170000	17000
HUNTER	0.0001	1000000+	370000	37000
FISHERMAN	0.0001	1000000+	410000	41000
RESIDENT	0.0001	1000000+	170000	17000

^aThe plus sign (+) means "greater than."

Table C-78

Margins of Safety for Exposed Members of the Public
 Aerial Routine-Worst Case Scenario
 Dicamba Sprayed on 400 acres by Fixed Wing

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (757.0)	SYSTEMIC NOEL (25.00)	REPRODUCTIVE NOEL (2.50)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0085	89000	2900	290
VEGETATION CONTACT				
HIKER	0.0001	1000000+	210000	21000
PICKER	0.0219	35000	1100	110
DRINKING WATER	0.0127	60000	2000	200
EATING BERRIES	0.0104	73000	2400	240
EATING VEGETS.	0.0208	36000	60	120
EATING DEER	0.0016	480000	16000	1600
EATING BIRD	0.0067	110000	3700	370
EATING FISH	0.0051	150000	4900	490
<u>For Combined Routes of Exposure</u>				
HIKER	0.0213	36000	1200	120
BERRY PICKER	0.0535	14000	470	47
HUNTER	0.0296	26000	850	85
FISHERMAN	0.0264	29000	950	95
RESIDENT	0.0421	18000	590	59

^aThe plus sign (+) means "greater than."

Table C-79

Margins of Safety for Exposed Members of the Public
 Backpack Routine-Worst Case Scenario
 Dicamba Sprayed on 60 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (757.0)	SYSTEMIC NOEL (25.00)	REPRODUCTIVE NOEL (2.50)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0003	1000000+	73000	7300
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	510000
PICKER	0.0009	860000	28000	2800
DRINKING WATER	0.0004	1000000+	65000	6500
EATING BERRIES	0.0007	1000000	34000	3400
EATING VEGETS.	0.0015	510000	17000	1700
EATING DEER	0.0001	1000000+	260000	26000
EATING BIRD	0.0003	1000000+	87000	8700
EATING FISH	0.0002	1000000+	160000	16000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0007	1000000	34000	3400
BERRY PICKER	0.0023	320000	11000	1100
HUNTER	0.0011	680000	22000	2200
FISHERMAN	0.0009	860000	28000	2800
RESIDENT	0.0022	340000	11000	1100

^aThe plus sign (+) means "greater than."

Table C-80

Margins of Safety for Exposed Members of the Public
 Right of Way Routine-Worst Case Scenario
 Dicamba Sprayed on 40 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (757.0)	SYSTEMIC NOEL (25.00)	REPRODUCTIVE NOEL (2.50)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0001	1000000+	230000	23000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0003	1000000+	90000	9000
DRINKING WATER	0.0002	1000000+	140000	14000
EATING BERRIES	0.0003	1000000+	90000	9000
EATING VEGETS.	0.0006	1000000+	45000	4500
EATING DEER	0.0000	1000000+	720000	72000
EATING BIRD	0.0001	1000000+	270000	27000
EATING FISH	0.0001	1000000+	350000	35000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0003	1000000+	87000	8700
BERRY PICKER	0.0008	900000	30000	3000
HUNTER	0.0004	1000000+	60000	6000
FISHERMAN	0.0004	1000000+	69000	6900
RESIDENT	0.0008	890000	30000	3000

^aThe plus sign (+) means "greater than."

Table C-81

Margins of Safety for Exposed Members of the Public
 Aerial Routine-Realistic Scenario
 Diuron Sprayed on 40 acres by Helicopter

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE	LD50	SYSTEMIC	REPRODUCTIVE
	(MG/KG/DAY)	(3750.0)	NOEL (0.63)	NOEL (6.25)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	----	----	----	----
VEGETATION CONTACT				
HIKER	----	----	----	----
PICKER	----	----	----	----
DRINKING WATER	----	----	----	----
EATING BERRIES	----	----	----	----
EATING VEGETS.	----	----	----	----
EATING DEER	----	----	----	----
EATING BIRD	----	----	----	----
EATING FISH	----	----	----	----
<u>For Combined Routes of Exposure</u>				
HIKER	----	----	----	----
BERRY PICKER	----	----	----	----
HUNTER	----	----	----	----
FISHERMAN	----	----	----	----
RESIDENT	----	----	----	----

Table C-82

Margins of Safety for Exposed Members of the Public
Backpack Routine-Realistic Scenario
Diuron Sprayed on 6 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (3750.0)	SYSTEMIC NOEL (0.63)	REPRODUCTIVE NOEL (6.25)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0004	1000000+	1800	18000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	120000	1000000+
PICKER	0.0014	1000000+	440	4400
DRINKING WATER	0.0003	1000000+	1900	19000
EATING BERRIES	0.0006	1000000+	1000	10000
EATING VEGETS.	0.0012	1000000+	510	5100
EATING DEER	0.0001	1000000+	7800	78000
EATING BIRD	0.0002	1000000+	2600	26000
EATING FISH	0.0027	1000000+	240	2400
<u>For Combined Routes of Exposure</u>				
HIKER	0.0007	1000000+	900	9000
BERRY PICKER	0.0027	1000000+	230	2300
HUNTER	0.0010	1000000+	610	6100
FISHERMAN	0.0033	1000000+	190	1900
RESIDENT	0.0019	1000000+	330	3300

^aThe plus sign (+) means "greater than."

Table C-83

Margins of Safety for Exposed Members of the Public
 Right-of-Way Routine-Realistic Scenario
 Diuron Sprayed on 12 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (3750.0)	SYSTEMIC NOEL (0.63)	REPRODUCTIVE NOEL (6.25)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0001	1000000+	9100	91000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	630000	1000000+
PICKER	0.0004	1000000+	1700	17000
DRINKING WATER	0.0001	1000000+	4200	42000
EATING BERRIES	0.0002	1000000+	3100	31000
EATING VEGETS.	0.0004	1000000+	1500	15000
EATING DEER	0.0000	1000000+	25000	250000
EATING BIRD	0.0001	1000000+	9200	92000
EATING FISH	0.0012	1000000+	520	5200
<u>For Combined Routes of Exposure</u>				
HIKER	0.0002	1000000+	2900	29000
BERRY PICKER	0.0008	1000000+	790	7900
HUNTER	0.0003	1000000+	2000	20000
FISHERMAN	0.0014	1000000+	440	4400
RESIDENT	0.0006	1000000+	1000	10000

^aThe plus sign (+) means "greater than."

Table C-84

Margins of Safety for Exposed Members of the Public
 Aerial Routine-Worst Case Scenario
 Diuron Sprayed on 400 acres by Fixed Wing

MARGIN OF SAFETY RELATIVE TO				
EXPOSURE (MG/KG/DAY)	LD50 (3750.0)	SYSTEMIC NOEL (0.63)	REPRODUCTIVE NOEL (6.25)	
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	---	---	---	---
VEGETATION CONTACT				
HIKER	---	---	---	---
PICKER	---	---	---	---
DRINKING WATER	---	---	---	---
EATING BERRIES	---	---	---	---
EATING VEGETS.	---	---	---	---
EATING DEER	---	---	---	---
EATING BIRD	---	---	---	---
EATING FISH	---	---	---	---
<u>For Combined Routes of Exposure</u>				
HIKER	---	---	---	---
BERRY PICKER	---	---	---	---
HUNTER	---	---	---	---
FISHERMAN	---	---	---	---
RESIDENT	---	---	---	---

Table C-85

Margins of Safety for Exposed Members of the Public
 Backpack Routine-Worst Case Scenario
 Diuron Sprayed on 60 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (3750.0)	SYSTEMIC NOEL (0.63)	REPRODUCTIVE NOEL (6.25)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0010	1000000+	610	6100
VEGETATION CONTACT				
HIKER	0.0000	1000000+	42000	420000
PICKER	0.0026	1000000+	240	2400
DRINKING WATER	0.0006	1000000+	1100	11000
EATING BERRIES	0.0011	1000000+	570	5700
EATING VEGETS.	0.0022	1000000+	280	2800
EATING DEER	0.0001	1000000+	4300	43000
EATING BIRD	0.0005	1000000+	1400	14000
EATING FISH	0.0046	820000	140	1400
<u>For Combined Routes of Exposure</u>				
HIKER	0.0016	1000000+	390	3900
BERRY PICKER	0.0054	700000	120	1200
HUNTER	0.0022	1000000+	280	2800
FISHERMAN	0.0062	600000	100	1000
RESIDENT	0.0038	980000	160	1600

^aThe plus sign (+) means "greater than."

Table C-86

Margins of Safety for Exposed Members of the Public
 Right of Way Routine-Worst Case Scenario
 Diuron Sprayed on 40 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (3750.0)	SYSTEMIC NOEL (0.63)	REPRODUCTIVE NOEL (6.25)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0010	1000000+	650	6500
VEGETATION CONTACT				
HIKER	0.0000	1000000+	45000	450000
PICKER	0.0025	1000000+	250	2500
DRINKING WATER	0.0008	1000000+	780	7800
EATING BERRIES	0.0012	1000000+	500	5000
EATING VEGETS.	0.0025	1000000+	250	2500
EATING DEER	0.0002	1000000+	4000	40000
EATING BIRD	0.0004	1000000+	1400	14000
EATING FISH	0.0064	590000	98	980
<u>For Combined Routes of Exposure</u>				
HIKER	0.0018	1000000+	350	3500
BERRY PICKER	0.0055	690000	110	1100
HUNTER	0.0024	1000000+	260	2600
FISHERMAN	0.0081	460000	77	770
RESIDENT	0.0043	880000	150	1500

^aThe plus sign (+) means "greater than."

Table C-87

Margins of Safety for Exposed Members of the Public
Aerial Routine-Realistic Scenario
Fosamine Sprayed on 40 acres by Helicopter

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (24400.0)	SYSTEMIC NOEL (25.00)	REPRODUCTIVE NOEL (500.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0000	1000000+	1000000	1000000+
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0049	1000000+	5100	100000
DRINKING WATER	0.0028	1000000+	8800	180000
EATING BERRIES	0.0016	1000000+	15000	310000
EATING VEGETS.	0.0032	1000000+	7700	150000
EATING DEER	0.0002	1000000+	110000	1000000+
EATING BIRD	0.0008	1000000+	30000	610000
EATING FISH	0.0011	1000000+	22000	440000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0029	1000000+	8800	170000
BERRY PICKER	0.0093	1000000+	2700	53000
HUNTER	0.0039	1000000+	6400	130000
FISHERMAN	0.0040	1000000+	6300	130000
RESIDENT	0.0061	1000000+	4100	80000

^aThe plus sign (+) means "greater than."

Table C-88

Margins of Safety for Exposed Members of the Public
Backpack Routine-Realistic Scenario
Fosamine Sprayed on 6 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (24400.0)	SYSTEMIC NOEL (25.00)	REPRODUCTIVE NOEL (500.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0003	1000000+	94000	1000000+
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0011	1000000+	24000	740000
DRINKING WATER	0.0002	1000000+	100000	1000000+
EATING BERRIES	0.0005	1000000+	54000	1000000
EATING VEGETS.	0.0009	1000000+	27000	850000
EATING DEER	0.0001	1000000+	420000	1000000+
EATING BIRD	0.0002	1000000+	140000	1000000+
EATING FISH	0.0001	1000000+	250000	1000000+
<u>For Combined Routes of Exposure</u>				
HIKER	0.0005	1000000+	48000	1000000
BERRY PICKER	0.0020	1000000+	12000	380000
HUNTER	0.0008	1000000+	33000	1000000+
FISHERMAN	0.0006	1000000+	40000	830000
RESIDENT	0.0014	1000000+	17000	540000

^aThe plus sign (+) means "greater than."

Table C-89

Margins of Safety for Exposed Members of the Public
 Right-of-Way Routine-Realistic Scenario
 Fosamine Sprayed on 12 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (24400.0)	SYSTEMIC NOEL (25.00)	REPRODUCTIVE NOEL (500.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0001	1000000+	360000	1000000+
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0004	1000000+	67000	1000000+
DRINKING WATER	0.0001	1000000+	170000	1000000+
EATING BERRIES	0.0002	1000000+	120000	1000000+
EATING VEGETS.	0.0004	1000000+	61000	1000000+
EATING DEER	0.0000	1000000+	980000	1000000+
EATING BIRD	0.0001	1000000+	370000	1000000+
EATING FISH	0.0001	1000000+	420000	1000000+
<u>For Combined Routes of Exposure</u>				
HIKER	0.0002	1000000+	110000	1000000+
BERRY PICKER	0.0008	1000000+	31000	630000
HUNTER	0.0003	1000000+	80000	1000000+
FISHERMAN	0.0003	1000000+	90000	1000000+
RESIDENT	0.0006	1000000+	40000	83000

^aThe plus sign (+) means "greater than."

Table C-90

Margins of Safety for Exposed Members of the Public
 Aerial Routine-Worst Case Scenario
 Fosamine Sprayed on 400 acres by Fixed Wing

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (24400.0)	SYSTEMIC NOEL (25.00)	REPRODUCTIVE NOEL (500.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0509	480000	490	10000
VEGETATION CONTACT				
HIKER	0.0007	1000000+	34000	670000
PICKER	0.1313	190000	190	3800
DRINKING WATER	0.0380	640000	660	13000
EATING BERRIES	0.0313	780000	800	16000
EATING VEGETS.	0.0625	390000	400	8000
EATING DEER	0.0049	1000000+	5100	100000
EATING BIRD	0.0213	1000000+	1200	23000
EATING FISH	0.0152	1000000+	1600	33000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0897	270000	280	5600
BERRY PICKER	0.2516	97000	99	2000
HUNTER	0.1159	210000	220	4300
FISHERMAN	0.1049	230000	240	4700
RESIDENT	0.1523	160000	160	3300

^aThe plus sign (+) means "greater than."

Table C-91

Margins of Safety for Exposed Members of the Public
 Backpack Routine-Worst Case Scenario
 Fosamine Sprayed on 60 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (24400.0)	SYSTEMIC NOEL (25.00)	REPRODUCTIVE NOEL (500.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0020	1000000+	13000	250000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	880000	1000000+
PICKER	0.0051	1000000+	4900	100000
DRINKING WATER	0.0011	1000000+	23000	450000
EATING BERRIES	0.0021	1000000+	12000	230000
EATING VEGETS.	0.0042	1000000+	5900	120000
EATING DEER	0.0003	1000000+	90000	1000000+
EATING BIRD	0.0009	1000000+	29000	570000
EATING FISH	0.0004	1000000+	57000	1000000+
<u>For Combined Routes of Exposure</u>				
HIKER	0.0031	1000000+	8100	160000
BERRY PICKER	0.0103	1000000+	2400	490000
HUNTER	0.0043	1000000+	5900	120000
FISHERMAN	0.0035	1000000+	7100	140000
RESIDENT	0.0073	1000000+	3400	67000

^aThe plus sign (+) means "greater than."

Table C-92

Margins of Safety for Exposed Members of the Public
 Right of Way Routine-Worst Case Scenario
 Fosamine Sprayed on 40 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (24400.0)	SYSTEMIC NOEL (25.00)	REPRODUCTIVE NOEL (500.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0006	1000000+	39000	830000+
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0017	1000000+	15000	300000
DRINKING WATER	0.0005	1000000+	47000	1000000+
EATING BERRIES	0.0008	1000000+	30000	600000
EATING VEGETS.	0.0017	1000000+	15000	300000
EATING DEER	0.0001	1000000+	240000	1000000+
EATING BIRD	0.0003	1000000+	85000	1000000+
EATING FISH	0.0002	1000000+	120000	1000000+
<u>For Combined Routes of Exposure</u>				
HIKER	0.0012	1000000+	21000	420000
BERRY PICKER	0.0037	1000000+	6800	140000
HUNTER	0.0016	1000000+	16000	310000
FISHERMAN	0.0014	1000000+	18000	360000
RESIDENT	0.0028	1000000+	8800	170000

^aThe plus sign (+) means "greater than."

Table C-93

Margins of Safety for Exposed Members of the Public
 Aerial Routine-Realistic Scenario
 Glyphosate Sprayed on 40 acres by Helicopter

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (4320.0)	SYSTEMIC NOEL (31.00)	REPRODUCTIVE NOEL (10.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0000	1000000+	1000000+	620000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0032	1000000+	9600	3100
DRINKING WATER	0.0019	1000000+	17000	5300
EATING BERRIES	0.0011	1000000+	29000	9300
EATING VEGETS.	0.0022	1000000+	15000	4600
EATING DEER	0.0002	1000000+	200000	65000
EATING BIRD	0.0005	1000000+	57000	18000
EATING FISH	0.0008	1000000+	42000	13000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0019	1000000+	17000	5300
BERRY PICKER	0.0062	690000	5000	1600
HUNTER	0.0026	1000000+	12000	3800
FISHERMAN	0.0027	1000000+	11000	3800
RESIDENT	0.0041	1000000+	7700	2500

^aThe plus sign (+) means "greater than."

Table C-94

Margins of Safety for Exposed Members of the Public
Backpack Routine-Realistic Scenario
Glyphosate Sprayed on 6 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (4320.0)	SYSTEMIC NOEL (31.00)	REPRODUCTIVE NOEL (10.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0001	1000000+	230000	75000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0005	1000000+	59000	19000
DRINKING WATER	0.0001	1000000+	250000	80000
EATING BERRIES	0.0002	1000000+	140000	44000
EATING VEGETS.	0.0005	1000000+	68000	22000
EATING DEER	0.0000	1000000+	1000000+	330000
EATING BIRD	0.0001	1000000+	340000	110000
EATING FISH	0.0000	1000000+	620000	200000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0003	1000000+	110000	38000
BERRY PICKER	0.0010	1000000+	31000	9800
HUNTER	0.0004	1000000+	82000	26000
FISHERMAN	0.0003	1000000+	100000	32000
RESIDENT	0.0007	1000000+	44000	14000

^aThe plus sign (+) means "greater than."

Table C-95

Margins of Safety for Exposed Members of the Public
 Right-of-Way Routine-Realistic Scenario
 Glyphosate Sprayed on 12 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (4320.0)	SYSTEMIC NOEL (31.00)	REPRODUCTIVE NOEL (10.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0000	1000000+	900000	290000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0002	1000000+	170000	54000
DRINKING WATER	0.0001	1000000+	420000	130000
EATING BERRIES	0.0001	1000000+	300000	98000
EATING VEGETS.	0.0002	1000000+	160000	49000
EATING DEER	0.0000	1000000+	1000000+	780000
EATING BIRD	0.0000	1000000+	930000	300000
EATING FISH	0.0000	1000000+	1000000+	340000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0001	1000000+	280000	91000
BERRY PICKER	0.0004	1000000+	78000	25000
HUNTER	0.0002	1000000+	200000	64000
FISHERMAN	0.0001	1000000+	230000	72000
RESIDENT	0.0003	1000000+	99000	32000

^aThe plus sign (+) means "greater than."

Table C-96

Margins of Safety for Exposed Members of the Public
 Aerial Routine-Worst Case Scenario
 Glyphosate Sprayed on 400 acres by Fixed Wing

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (4320.0)	SYSTEMIC NOEL (31.00)	REPRODUCTIVE NOEL (10.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0212	200000	1500	470
VEGETATION CONTACT				
HIKER	0.0003	1000000+	100000	33000
PICKER	0.0547	79000	570	180
DRINKING WATER	0.0159	270000	2000	630
EATING BERRIES	0.0130	330000	2400	770
EATING VEGETS.	0.0261	170000	1200	380
EATING DEER	0.0020	1000000+	16000	4900
EATING BIRD	0.0089	490000	3500	1100
EATING FISH	0.0063	680000	4900	1600
<u>For Combined Routes of Exposure</u>				
HIKER	0.0374	120000	830	270
BERRY PICKER	0.1048	41000	300	95
HUNTER	0.0483	89000	640	210
FISHERMAN	0.0437	99000	720	230
RESIDENT	0.0634	68000	490	160

^aThe plus sign (+) means "greater than."

Table C-97

Margins of Safety for Exposed Members of the Public
 Backpack Routine-Worst Case Scenario
 Glyphosate Sprayed on 60 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (4320.0)	SYSTEMIC NOEL (31.00)	REPRODUCTIVE NOEL (10.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0009	1000000+	36000	12000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	810000
PICKER	0.0022	1000000+	15000	4500
DRINKING WATER	0.0005	1000000+	66000	21000
EATING BERRIES	0.0009	1000000+	34000	11000
EATING VEGETS.	0.0018	1000000+	17000	5400
EATING DEER	0.0001	1000000+	260000	83000
ATING BIRD	0.0004	1000000+	82000	26000
EATING FISH	0.0002	1000000+	170000	52000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0013	1000000+	23000	7400
BERRY PICKER	0.0045	970000	7000	2200
HUNTER	0.0018	1000000+	17000	5400
FISHERMAN	0.0015	1000000+	21000	6500
RESIDENT	0.0032	1000000+	9800	3100

^aThe plus sign (+) means "greater than."

Table C-98

Margins of Safety for Exposed Members of the Public
 Right of Way Routine-Worst Case Scenario
 Glyphosate Sprayed on 40 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (4320.0)	SYSTEMIC NOEL (31.00)	REPRODUCTIVE NOEL (10.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0003	1000000+	110000	33000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0008	1000000+	41000	13000
DRINKING WATER	0.0002	1000000+	130000	40000
EATING BERRIES	0.0004	1000000+	80000	26000
EATING VEGETS.	0.0008	1000000+	41000	13000
EATING DEER	0.0000	1000000+	630000	200000
EATING BIRD	0.0001	1000000+	230000	73000
EATING FISH	0.0001	1000000+	310000	100000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0006	1000000+	56000	18000
BERRY PICKER	0.0017	1000000+	19000	5900
HUNTER	0.0007	1000000+	43000	14000
FISHERMAN	0.0007	1000000+	48000	15000
RESIDENT	0.0013	1000000+	24000	7500

^aThe plus sign (+) means "greater than."

Table C-99

Margins of Safety for Exposed Members of the Public
 Aerial Routine-Realistic Scenario
 Hexazinone Sprayed on 40 acres by Helicopter

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (1690.0)	SYSTEMIC NOEL (10.00)	REPRODUCTIVE NOEL (125.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0000	1000000+	500000	1000000+
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0041	420000	2500	31000
DRINKING WATER	0.0024	720000	4200	53000
EATING BERRIES	0.0013	1000000+	7400	93000
EATING VEGETS.	0.0027	630000	3700	46000
EATING DEER	0.0002	1000000+	52000	650000
EATING BIRD	0.0007	1000000+	15000	180000
EATING FISH	0.0009	1000000+	11000	130000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0024	710000	4200	53000
BERRY PICKER	0.0078	220000	1300	16000
HUNTER	0.0033	520000	3100	38000
FISHERMAN	0.0033	510000	3000	38000
RESIDENT	0.0051	330000	2000	25000

^aThe plus sign (+) means "greater than."

Table C-100

Margins of Safety for Exposed Members of the Public
 Backpack Routine-Realistic Scenario
 Hexazinone Sprayed on 6 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (1690.0)	SYSTEMIC NOEL (10.00)	REPRODUCTIVE NOEL (125.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0001	1000000+	100000	1000000+
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0004	1000000+	25000	320000
DRINKING WATER	0.0001	1000000+	110000	1000000+
EATING BERRIES	0.0002	1000000+	58000	730000
EATING VEGETS.	0.0003	1000000+	29000	360000
EATING DEER	0.0000	1000000+	450000	1000000+
EATING BIRD	0.0001	1000000+	150000	1000000+
EATING FISH	0.0000	1000000+	270000	1000000+
<u>For Combined Routes of Exposure</u>				
HIKER	0.0002	1000000+	52000	640000
BERRY PICKER	0.0008	1000000+	13000	160000
HUNTER	0.0003	1000000+	35000	440000
FISHERMAN	0.0002	1000000+	43000	540000
RESIDENT	0.0005	1000000+	19000	230000

^aThe plus sign (+) means "greater than."

Table C-101

Margins of Safety for Exposed Members of the Public
 Right-of-Way Routine-Realistic Scenario
 Hexazinone Sprayed on 12 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (1690.0)	SYSTEMIC NOEL (10.00)	REPRODUCTIVE NOEL (125.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0000	1000000+	230000	1000000+
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0002	1000000+	43000	540000
DRINKING WATER	0.0001	1000000+	110000	1000000+
EATING BERRIES	0.0001	1000000+	78000	980000
EATING VEGETS.	0.0003	1000000+	39000	490000
EATING DEER	0.0000	1000000+	630000	1000000+
EATING BIRD	0.0000	1000000+	240000	1000000+
EATING FISH	0.0000	1000000+	270000	1000000+
<u>For Combined Routes of Exposure</u>				
HIKER	0.0001	1000000+	73000	910000
BERRY PICKER	0.0005	1000000+	20000	250000
HUNTER	0.0002	1000000+	51000	640000
FISHERMAN	0.0002	1000000+	57000	720000
RESIDENT	0.0004	1000000+	25000	320000

^aThe plus sign (+) means "greater than."

Table C-102

Margins of Safety for Exposed Members of the Public
 Aerial Routine-Worst Case Scenario
 Hexazinone Sprayed on 400 acres by Fixed Wing

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (1690.0)	SYSTEMIC NOEL (10.00)	REPRODUCTIVE NOEL (125.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0127	130000	790	9800
VEGETATION CONTACT				
HIKER	0.0002	1000000+	55000	680000
PICKER	0.0328	51000	300	3800
DRINKING WATER	0.0095	180000	1100	13000
EATING BERRIES	0.0078	220000	1300	16000
EATING VEGETS.	0.0156	110000	640	8000
EATING DEER	0.0012	1000000+	8200	100000
EATING BIRD	0.0053	320000	1900	23000
EATING FISH	0.0038	440000	2600	33000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0224	75000	450	5600
BERRY PICKER	0.0629	27000	160	2000
HUNTER	0.0290	58000	350	4300
FISHERMAN	0.0262	64000	380	4800
RESIDENT	0.0381	44000	260	3300

^aThe plus sign (+) means "greater than."

Table C-103

Margins of Safety for Exposed Members of the Public
Backpack Routine-Worst Case Scenario
Hexazinone Sprayed on 60 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (1690.0)	SYSTEMIC NOEL (10.00)	REPRODUCTIVE NOEL (125.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0005	1000000+	19000	240000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0013	1000000+	7500	94000
DRINKING WATER	0.0003	1000000+	35000	440000
EATING BERRIES	0.0006	1000000+	18000	230000
EATING VEGETS.	0.0011	1000000+	9000	110000
EATING DEER	0.0001	1000000+	140000	1000000+
EATING BIRD	0.0002	1000000+	44000	550000
EATING FISH	0.0001	1000000+	87000	1000000+
<u>For Combined Routes of Exposure</u>				
HIKER	0.0008	1000000+	12000	150000
BERRY PICKER	0.0027	630000	3700	47000
HUNTER	0.0011	1000000+	9000	110000
FISHERMAN	0.0009	1000000+	11000	140000
RESIDENT	0.0019	880000	5200	65000

^aThe plus sign (+) means "greater than."

Table C-104

Margins of Safety for Exposed Members of the Public
 Right of Way Routine-Worst Case Scenario
 Hexazinone Sprayed on 40 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (1690.0)	SYSTEMIC NOEL (10.00)	REPRODUCTIVE NOEL (125.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0004	1000000+	28000	350000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0009	1000000+	11000	130000
DRINKING WATER	0.0003	1000000+	33000	420000
EATING BERRIES	0.0005	1000000+	21000	270000
EATING VEGETS.	0.0009	1000000+	11000	130000
EATING DEER	0.0001	1000000+	170000	1000000+
EATING BIRD	0.0002	1000000+	61000	760000
EATING FISH	0.0001	1000000+	84000	1000000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0007	1000000+	15000	190000
BERRY PICKER	0.0021	820000	4900	61000
HUNTER	0.0009	1000000+	11000	140000
FISHERMAN	0.0008	1000000+	13000	160000
RESIDENT	0.0016	1000000+	6300	78000

^aThe plus sign (+) means "greater than."

Table C-105

Margins of Safety for Exposed Members of the Public
 Aerial Routine-Realistic Scenario
 Picloram Sprayed on 40 acres by Helicopter

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (8200.0)	SYSTEMIC NOEL (7.00)	REPRODUCTIVE NOEL (50.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0000	1000000+	1000000+	1000000+
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0001	1000000+	90000	640000
DRINKING WATER	0.0009	1000000+	7400	53000
EATING BERRIES	0.0005	1000000+	13000	93000
EATING VEGETS.	0.0011	1000000+	6500	46000
EATING DEER	0.0001	1000000+	96000	680000
EATING BIRD	0.0002	1000000+	29000	200000
EATING FISH	0.0004	1000000+	19000	130000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0009	1000000+	7400	53000
BERRY PICKER	0.0016	1000000+	4500	32000
HUNTER	0.0013	1000000+	5600	40000
FISHERMAN	0.0013	1000000+	5300	38000
RESIDENT	0.0020	1000000+	3500	25000

^aThe plus sign (+) means "greater than."

Table C-106

Margins of Safety for Exposed Members of the Public
 Backpack Routine-Realistic Scenario
 Picloram Sprayed on 6 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (8200.0)	SYSTEMIC NOEL (7.00)	REPRODUCTIVE NOEL (50.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0000	1000000+	1000000+	1000000+
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0000	1000000+	410000	1000000+
DRINKING WATER	0.0001	1000000+	84000	600000
EATING BERRIES	0.0002	1000000+	46000	330000
EATING VEGETS.	0.0003	1000000+	23000	160000
EATING DEER	0.0000	1000000+	370000	1000000+
EATING BIRD	0.0001	1000000+	130000	910000
EATING FISH	0.0000	1000000+	210000	1000000+
<u>For Combined Routes of Exposure</u>				
HIKER	0.0001	1000000+	80000	570000
BERRY PICKER	0.0003	1000000+	27000	190000
HUNTER	0.0002	1000000+	43000	310000
FISHERMAN	0.0001	1000000+	58000	420000
RESIDENT	0.0004	1000000+	18000	130000

^aThe plus sign (+) means "greater than."

Table C-107

Margins of Safety for Exposed Members of the Public
 Right-of-Way Routine-Realistic Scenario
 Picloram Sprayed on 12 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (8200.0)	SYSTEMIC NOEL (7.00)	REPRODUCTIVE NOEL (50.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0000	1000000+	1000000+	1000000+
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0000	1000000+	1000000+	1000000+
DRINKING WATER	0.0000	1000000+	190000	1000000+
EATING BERRIES	0.0001	1000000+	140000	980000
EATING VEGETS.	0.0001	1000000+	68000	490000
EATING DEER	0.0000	1000000+	1000000+	1000000+
EATING BIRD	0.0000	1000000+	460000	1000000+
EATING FISH	0.0000	1000000+	470000	1000000+
<u>For Combined Routes of Exposure</u>				
HIKER	0.0000	1000000+	180000	1000000+
BERRY PICKER	0.0001	1000000+	75000	530000
HUNTER	0.0001	1000000+	120000	840000
FISHERMAN	0.0001	1000000+	130000	940000
RESIDENT	0.0001	1000000+	50000	360000

^aThe plus sign (+) means "greater than."

Table C-108

Margins of Safety for Exposed Members of the Public
Aerial Routine-Worst Case Scenario
Picloram Sprayed on 400 acres by Fixed Wing

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (8200.0)	SYSTEMIC NOEL (7.00)	REPRODUCTIVE NOEL (50.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0010	1000000+	6900	49000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	480000	1000000+
PICKER	0.0026	1000000+	2700	19000
DRINKING WATER	0.0159	520000	440	3200
EATING BERRIES	0.0130	630000	540	3800
EATING VEGETS.	0.0261	310000	270	1900
EATING DEER	0.0019	1000000+	3700	26000
EATING BIRD	0.0079	1000000	890	6300
EATING FISH	0.0063	1000000+	1100	7900
<u>For Combined Routes of Exposure</u>				
HIKER	0.0169	490000	410	3000
BERRY PICKER	0.0325	250000	220	1500
HUNTER	0.0267	310000	260	1900
FISHERMAN	0.0232	350000	300	2200
RESIDENT	0.0429	190000	160	1200

^aThe plus sign (+) means "greater than."

Table C-109

Margins of Safety for Exposed Members of the Public
 Backpack Routine-Worst Case Scenario
 Picloram Sprayed on 60 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (8200.0)	SYSTEMIC NOEL (7.00)	REPRODUCTIVE NOEL (50.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0000	1000000+	210000	1000000+
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0001	1000000+	83000	590000
DRINKING WATER	0.0004	1000000+	18000	130000
EATING BERRIES	0.0007	1000000+	9500	68000
EATING VEGETS.	0.0015	1000000+	4700	34000
EATING DEER	0.0001	1000000+	76000	540000
EATING BIRD	0.0003	1000000+	26000	180000
EATING FISH	0.0002	1000000+	46000	330000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0004	1000000+	17000	120000
BERRY PICKER	0.0012	1000000+	5700	40000
HUNTER	0.0008	1000000+	9000	64000
FISHERMAN	0.0006	1000000+	12000	88000
RESIDENT	0.0019	1000000+	3700	26000

^aThe plus sign (+) means "greater than."

Table C-110

Margins of Safety for Exposed Members of the Public
 Right of Way Routine-Worst Case Scenario
 Picloram Sprayed on 40 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (8200.0)	SYSTEMIC NOEL (7.00)	REPRODUCTIVE NOEL (50.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0000	1000000+	1000000+	1000000+
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0000	1000000+	470000	1000000+
DRINKING WATER	0.0001	1000000+	70000	500000
EATING BERRIES	0.0002	1000000+	45000	320000
EATING VEGETS.	0.0003	1000000+	23000	160000
EATING DEER	0.0000	1000000+	370000	1000000+
EATING BIRD	0.0000	1000000+	140000	1000000
EATING FISH	0.0000	1000000+	180000	1000000+
<u>For Combined Routes of Exposure:</u>				
HIKER	0.0001	1000000+	66000	470000
BERRY PICKER	0.0003	1000000+	25000	180000
HUNTER	0.0002	1000000+	40000	290000
FISHERMAN	0.0001	1000000+	48000	340000
RESIDENT	0.0004	1000000+	17000	120000

^aThe plus sign (+) means "greater than."

Table C-111

Margins of Safety for Exposed Members of the Public
 Aerial Routine-Realistic Scenario
 Simazine Sprayed on 40 acres by Helicopter

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (5000.0)	SYSTEMIC NOEL (5.00)	REPRODUCTIVE NOEL (5.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0000	1000000+	160000	160000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0065	770000	770	770
DRINKING WATER	0.0038	1000000+	1300	1300
EATING BERRIES	0.0022	1000000+	2300	2300
EATING VEGETS.	0.0043	1000000+	1200	1200
EATING DEER	0.0003	1000000+	16000	16000
EATING BIRD	0.0011	1000000+	4500	4500
EATING FISH	0.0015	1000000+	3300	3300
<u>For Combined Routes of Exposure</u>				
HIKER	0.0038	1000000+	1300	1300
BERRY PICKER	0.0124	400000	400	400
HUNTER	0.0052	960000	960	960
FISHERMAN	0.0053	940000	940	940
RESIDENT	0.0081	620000	620	620

^aThe plus sign (+) means "greater than."

Table C-112

Margins of Safety for Exposed Members of the Public
Backpack Routine-Realistic Scenario
Simazine Sprayed on 6 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (5000.0)	SYSTEMIC NOEL (5.00)	REPRODUCTIVE NOEL (5.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0002	1000000+	28000	28000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0007	1000000+	7100	7100
DRINKING WATER	0.0002	1000000+	30000	30000
EATING BERRIES	0.0003	1000000+	16000	16000
EATING VEGETS.	0.0006	1000000+	8200	8200
EATING DEER	0.0000	1000000+	130000	130000
EATING BIRD	0.0001	1000000+	41000	41000
EATING FISH	0.0001	1000000+	75000	75000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0003	1000000+	14000	14000
BERRY PICKER	0.0014	1000000+	3700	3700
HUNTER	0.0005	1000000+	9800	9800
FISHERMAN	0.0004	1000000+	12000	12000
RESIDENT	0.0010	1000000+	5200	5200

^aThe plus sign (+) means "greater than."

Table C-113

Margins of Safety for Exposed Members of the Public
 Right-of-Way Routine-Realistic Scenario
 Simazine Sprayed on 12 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (5000.0)	SYSTEMIC NOEL (5.00)	REPRODUCTIVE NOEL (5.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0000	1000000+	140000	140000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0002	1000000+	27000	27000
DRINKING WATER	0.0001	1000000+	67000	67000
EATING BERRIES	0.0001	1000000+	49000	49000
EATING VEGETS.	0.0002	1000000+	24000	24000
EATING DEER	0.0000	1000000+	390000	390000
EATING BIRD	0.0000	1000000+	150000	150000
EATING FISH	0.0000	1000000+	170000	170000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0001	1000000+	46000	46000
BERRY PICKER	0.0004	1000000+	13000	13000
HUNTER	0.0002	1000000+	32000	32000
FISHERMAN	0.0001	1000000+	36000	36000
RESIDENT	0.0003	1000000+	16000	16000

^aThe plus sign (+) means "greater than."

Table C-114

Margins of Safety for Exposed Members of the Public
 Aerial Routine-Worst Case Scenario
 Simazine Sprayed on 400 acres by Fixed Wing

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (5000.0)	SYSTEMIC NOEL (5.00)	REPRODUCTIVE NOEL (5.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0212	240000	240	240
VEGETATION CONTACT				
HIKER	0.0003	1000000+	16000	16000
PICKER	0.0547	91000	91	91
DRINKING WATER	0.0159	320000	320	320
EATING BERRIES	0.0130	380000	380	380
EATING VEGETS.	0.0261	190000	190	190
EATING DEER	0.0020	1000000+	2500	2500
EATING BIRD	0.0089	560000	560	560
EATING FISH	0.0063	790000	790	790
<u>For Combined Routes of Exposure</u>				
HIKER	0.0374	130000	130	130
BERRY PICKER	0.1048	48000	48	48
HUNTER	0.0483	100000	100	100
FISHERMAN	0.0437	110000	110	110
RESIDENT	0.0634	79000	79	79

^aThe plus sign (+) means "greater than."

Table C-115

Margins of Safety for Exposed Members of the Public
 Backpack Routine-Worst Case Scenario
 Simazine Sprayed on 60 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (5000.0)	SYSTEMIC NOEL (5.00)	REPRODUCTIVE NOEL (5.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0008	1000000+	6300	6300
VEGETATION CONTACT				
HIKER	0.0000	1000000+	440000	440000
PICKER	0.0020	1000000+	2500	2500
DRINKING WATER	0.0004	1000000+	11000	11000
EATING BERRIES	0.0008	1000000+	5900	5900
EATING VEGETS.	0.0017	1000000+	2900	2900
EATING DEER	0.0001	1000000+	45000	45000
EATING BIRD	0.0004	1000000+	14000	14000
EATING FISH	0.0002	1000000+	28000	28000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0012	1000000+	4000	4000
BERRY PICKER	0.0041	1000000+	1200	1200
HUNTER	0.0017	1000000+	2900	2900
FISHERMAN	0.0014	1000000+	3500	3500
RESIDENT	0.0029	1000000+	1700	1700

^aThe plus sign (+) means "greater than."

Table C-116

Margins of Safety for Exposed Members of the Public
 Right of Way Routine-Worst Case Scenario
 Simazine Sprayed on 40 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (5000.0)	SYSTEMIC NOEL (5.00)	REPRODUCTIVE NOEL (5.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0003	1000000+	18000	18000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0007	1000000+	7000	7000
DRINKING WATER	0.0002	1000000+	22000	22000
EATING BERRIES	0.0004	1000000+	14000	14000
EATING VEGETS.	0.0007	1000000+	7000	7000
EATING DEER	0.0000	1000000+	110000	110000
EATING BIRD	0.0001	1000000+	39000	39000
EATING FISH	0.0001	1000000+	55000	55000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0005	1000000+	9800	9800
BERRY PICKER	0.0016	1000000+	3200	3200
HUNTER	0.0007	1000000+	7300	7300
FISHERMAN	0.0006	1000000+	8300	8300
RESIDENT	0.0012	1000000+	4100	4100

^aThe plus sign (+) means "greater than."

Table C-117

Margins of Safety for Exposed Members of the Public
 Aerial Routine-Realistic Scenario
 Tebuthiuron Sprayed on 40 acres by Helicopter

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (644.0)	SYSTEMIC NOEL (12.50)	REPRODUCTIVE NOEL (90.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0000	1000000+	1000000+	1000000+
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0016	400000	7700	55000
DRINKING WATER	0.0009	680000	13000	96000
EATING BERRIES	0.0005	1000000+	23000	170000
EATING VEGETS.	0.0011	600000	12000	83000
EATING DEER	0.0001	1000000+	160000	1000000+
EATING BIRD	0.0003	1000000+	45000	330000
EATING FISH	0.0038	170000	3300	24000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0010	680000	13000	95000
BERRY PICKER	0.0031	210000	4000	29000
HUNTER	0.0013	490000	9600	69000
FISHERMAN	0.0047	140000	2600	19000
RESIDENT	0.0020	320000	6200	44000

^aThe plus sign (+) means "greater than."

Table C-118

Margins of Safety for Exposed Members of the Public
Backpack Routine-Realistic Scenario
Tebuthiuron Sprayed on 6 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE	LD50	SYSTEMIC	REPRODUCTIVE
	(MG/KG/DAY)	(644.0)	NOEL (12.50)	NOEL (90.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0001	1000000+	94000	670000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0005	1000000+	24000	170000
DRINKING WATER	0.0001	1000000+	100000	720000
EATING BERRIES	0.0002	1000000+	54000	390000
EATING VEGETS.	0.0005	1000000+	27000	200000
EATING DEER	0.0000	1000000+	420000	1000000+
EATING BIRD	0.0001	1000000+	140000	980000
EATING FISH	0.0005	1000000+	25000	180000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0003	1000000+	48000	350000
BERRY PICKER	0.0010	630000	12000	89000
HUNTER	0.0004	1000000+	33000	240000
FISHERMAN	0.0008	850000	17000	120000
RESIDENT	0.0007	900000	17000	130000

^aThe plus sign (+) means "greater than."

Table C-119

Margins of Safety for Exposed Members of the Public
 Right-of-Way Routine-Realistic Scenario
 Tebuthiuron Sprayed on 12 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (644.0)	SYSTEMIC NOEL (12.50)	REPRODUCTIVE NOEL (90.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0000	1000000+	330000	1000000+
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0002	1000000+	61000	440000
DRINKING WATER	0.0001	1000000+	150000	1000000+
EATING BERRIES	0.0001	1000000+	110000	800000
EATING VEGETS.	0.0002	1000000+	56000	400000
EATING DEER	0.0000	1000000+	890000	1000000+
EATING BIRD	0.0000	1000000+	340000	1000000+
EATING FISH	0.0003	1000000+	38000	270000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0001	1000000+	100000	750000
BERRY PICKER	0.0004	1000000+	29000	210000
HUNTER	0.0002	1000000+	73000	520000
FISHERMAN	0.0004	1000000+	28000	200000
RESIDENT	0.0003	1000000+	36000	260000

^aThe plus sign (+) means "greater than."

Table C-120

Margins of Safety for Exposed Members of the Public
 Aerial Routine-Worst Case Scenario
 Tebuthiuron Sprayed on 400 acres by Fixed Wing

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (644.0)	SYSTEMIC NOEL (12.50)	REPRODUCTIVE NOEL (90.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0255	25000	490	3500
VEGETATION CONTACT				
HIKER	0.0004	1000000+	34000	250000
PICKER	0.0657	9800	190	1400
DRINKING WATER	0.0190	34000	660	4700
EATING BERRIES	0.0156	41000	800	5800
EATING VEGETS.	0.0313	21000	400	2900
EATING DEER	0.0024	260000	5100	37000
EATING BIRD	0.0107	60000	1200	8400
EATING FISH	0.0761	8500	160	1200
<u>For Combined Routes of Exposure</u>				
HIKER	0.0449	14000	280	2000
BERRY PICKER	0.1258	5100	99	720
HUNTER	0.0580	11000	220	1600
FISHERMAN	0.1209	5300	100	740
RESIDENT	0.0761	8500	160	1200

^aThe plus sign (+) means "greater than."

Table C-121

Margins of Safety for Exposed Members of the Public
 Backpack Routine-Worst Case Scenario
 Tebuthiuron Sprayed on 60 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (644.0)	SYSTEMIC NOEL (12.50)	REPRODUCTIVE NOEL (90.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0010	630000	12000	88000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	850000	1000000+
PICKER	0.0026	240000	4700	34000
DRINKING WATER	0.0006	1000000+	22000	160000
EATING BERRIES	0.0011	580000	11000	81000
EATING VEGETS.	0.0022	290000	5700	41000
EATING DEER	0.0001	1000000+	86000	620000
EATING BIRD	0.0005	1000000+	27000	200000
EATING FISH	0.0023	280000	5500	39000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0016	400000	7700	56000
BERRY PICKER	0.0054	120000	2300	17000
HUNTER	0.0022	290000	5600	41000
FISHERMAN	0.0039	160000	3200	23000
RESIDENT	0.0038	170000	3300	24000

^aThe plus sign (+) means "greater than."

Table C-122

Margins of Safety for Exposed Members of the Public
 Right of Way Routine-Worst Case Scenario
 Tebuthiuron Sprayed on 40 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (644.0)	SYSTEMIC NOEL (12.50)	REPRODUCTIVE NOEL (90.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0003	1000000+	45000	330000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0007	910000	18000	130000
DRINKING WATER	0.0002	1000000+	55000	390000
EATING BERRIES	0.0004	1000000+	35000	250000
EATING VEGETS.	0.0007	900000	18000	130000
EATING DEER	0.0000	1000000+	280000	1000000+
EATING BIRD	0.0001	1000000+	99000	710000
EATING FISH	0.0009	700000	14000	98000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0005	1000000+	25000	180000
BERRY PICKER	0.0016	410000	7900	57000
HUNTER	0.0007	950000	18000	130000
FISHERMAN	0.0014	450000	8800	63000
RESIDENT	0.0012	530000	10000	74000

^aThe plus sign (+) means "greater than."

Table C-123

Margins of Safety for Exposed Members of the Public
Aerial Routine-Realistic Scenario
Triclopyr Sprayed on 40 acres by Helicopter

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (630.0)	SYSTEMIC NOEL (2.50)	REPRODUCTIVE NOEL (10.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0000	1000000+	160000	620000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0032	190000	770	3100
DRINKING WATER	0.0019	330000	1300	5300
EATING BERRIES	0.0011	580000	2300	9300
EATING VEGETS.	0.0022	290000	1200	4600
EATING DEER	0.0002	1000000+	16000	65000
EATING BIRD	0.0005	1000000+	4500	18000
EATING FISH	0.0008	840000	3300	13000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0019	330000	1300	5300
BERRY PICKER	0.0062	100000	400	1600
HUNTER	0.0026	240000	960	3800
FISHERMAN	0.0027	240000	940	3800
RESIDENT	0.0041	160000	620	2500

^aThe plus sign (+) means "greater than."

Table C-124

Margins of Safety for Exposed Members of the Public
 Backpack Routine-Realistic Scenario
 Triclopyr Sprayed on 6 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (630.0)	SYSTEMIC NOEL (2.50)	REPRODUCTIVE NOEL (10.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0002	1000000+	14000	56000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	980000	1000000+
PICKER	0.0007	890000	3500	14000
DRINKING WATER	0.0002	1000000+	15000	60000
EATING BERRIES	0.0003	1000000+	8200	33000
EATING VEGETS.	0.0006	1000000	4100	16000
EATING DEER	0.0000	1000000+	63000	250000
EATING BIRD	0.0001	1000000+	20000	82000
EATING FISH	0.0001	1000000+	38000	150000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0003	1000000+	7200	29000
BERRY PICKER	0.0014	470000	1800	7400
HUNTER	0.0005	1000000+	4900	20000
FISHERMAN	0.0004	1000000+	6100	24000
RESIDENT	0.0010	660000	2600	10000

^aThe plus sign (+) means "greater than."

Table C-125

Margins of Safety for Exposed Members of the Public
 Right-of-Way Routine-Realistic Scenario
 Triclopyr Sprayed on 12 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (630.0)	SYSTEMIC NOEL (2.50)	REPRODUCTIVE NOEL (10.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0000	1000000+	72000	290000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	1000000+	1000000+
PICKER	0.0002	1000000+	13000	54000
DRINKING WATER	0.0001	1000000+	34000	130000
EATING BERRIES	0.0001	1000000+	24000	98000
EATING VEGETS.	0.0002	1000000+	12000	49000
EATING DEER	0.0000	1000000+	200000	780000
EATING BIRD	0.0000	1000000+	74000	300000
EATING FISH	0.0000	1000000+	84000	340000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0001	1000000+	23000	91000
BERRY PICKER	0.0004	1000000+	6300	25000
HUNTER	0.0002	1000000+	16000	64000
FISHERMAN	0.0001	1000000+	18000	72000
RESIDENT	0.0003	1000000+	8000	32000

^aThe plus sign (+) means "greater than."

Table C-126

Margins of Safety for Exposed Members of the Public
 Aerial Routine-Worst Case Scenario
 Triclopyr Sprayed on 400 acres by Fixed Wing

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (630.0)	SYSTEMIC NOEL (2.50)	REPRODUCTIVE NOEL (10.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0340	19000	74	290
VEGETATION CONTACT				
HIKER	0.0005	1000000+	5100	21000
PICKER	0.0876	7200	29	110
DRINKING WATER	0.0254	25000	99	390
EATING BERRIES	0.0208	30000	120	480
EATING VEGETS.	0.0417	15000	60	240
EATING DEER	0.0033	190000	770	3100
EATING BIRD	0.0142	44000	180	700
EATING FISH	0.0101	62000	250	990
<u>For Combined Routes of Exposure</u>				
HIKER	0.0598	11000	42	170
BERRY PICKER	0.1677	3800	15	60
HUNTER	0.0773	8200	32	130
FISHERMAN	0.0700	9000	36	140
RESIDENT	0.1015	6200	25	99

^aThe plus sign (+) means "greater than."

Table C-127

Margins of Safety for Exposed Members of the Public
 Backpack Routine-Worst Case Scenario
 Triclopyr Sprayed on 60 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (630.0)	SYSTEMIC NOEL (2.50)	REPRODUCTIVE NOEL (10.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0014	460000	1800	7300
VEGETATION CONTACT				
HIKER	0.0000	1000000+	130000	510000
PICKER	0.0035	180000	710	2800
DRINKING WATER	0.0008	820000	3300	13000
EATING BERRIES	0.0015	430000	1700	6800
EATING VEGETS.	0.0029	210000	850	3400
EATING DEER	0.0002	1000000+	13000	52000
EATING BIRD	0.0006	1000000	4100	16000
EATING FISH	0.0003	1000000+	8200	33000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0022	290000	1200	4600
BERRY PICKER	0.0071	88000	350	1400
HUNTER	0.0030	210000	850	3400
FISHERMAN	0.0025	260000	1000	4100
RESIDENT	0.0051	120000	490	2000

^aThe plus sign (+) means "greater than."

Table C-128

Margins of Safety for Exposed Members of the Public
 Right of Way Routine-Worst Case Scenario
 Triclopyr Sprayed on 40 acres

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (630.0)	SYSTEMIC NOEL (2.50)	REPRODUCTIVE NOEL (10.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0005	1000000+	5200	21000
VEGETATION CONTACT				
HIKER	0.0000	1000000+	360000	1000000+
PICKER	0.0012	510000	2000	8100
DRINKING WATER	0.0004	1000000+	6300	25000
EATING BERRIES	0.0006	1000000	4000	16000
EATING VEGETS.	0.0012	510000	2000	8100
EATING DEER	0.0001	1000000+	32000	130000
EATING BIRD	0.0002	1000000+	11000	45000
EATING FISH	0.0002	1000000+	16000	63000
<u>For Combined Routes of Exposure</u>				
HIKER	0.0009	710000	2800	11000
BERRY PICKER	0.0027	230000	910	3700
HUNTER	0.0012	530000	2100	8400
FISHERMAN	0.0010	600000	2400	9600
RESIDENT	0.0021	300000	1200	4700

^aThe plus sign (+) means "greater than."

Table C-129

Margins of Safety for Exposed Members of the Public
Accidental-Worst Case Spraying with Amitrole

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (4080.0)	SYSTEMIC NOEL (0.03)	REPRODUCTIVE NOEL (5.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0033	1000000+	7.5	1500
VEGETATION CONTACT				
HIKER	0.0000	1000000+	520	100000
PICKER	0.0086	470000	2.9	580
DRINKING WATER	0.1174	35000	-4.7	43
EATING BERRIES	0.0932	44000	-3.7	54
EATING VEGETS.	0.1935	21000	-7.7	26
EATING DEER	0.0187	220000	1.3	270
EATING BIRD	0.1161	35000	-4.6	43
EATING FISH	0.0470	87000	-1.9	110
<u>For Combined Routes of Exposure:</u>				
HIKER	0.1208	34000	-4.8	41
BERRY PICKER	0.2225	18000	-8.9	22
HUNTER	0.2556	16000	-10	20
FISHERMAN	0.1677	24000	-6.7	30
RESIDENT	0.3143	13000	-13	16

^aThe plus sign (+) means "greater than."

Table C-130

Margins of Safety for Exposed Members of the Public
Accidental-Worst Case Spraying with Asulam

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (4000.0)	SYSTEMIC NOEL (50.00)	REPRODUCTIVE NOEL (50.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.2085	19000	240	240
VEGETATION CONTACT				
HIKER	0.0030	1000000+	17000	17000
PICKER	0.5376	7400	93	93
DRINKING WATER	0.0734	55000	680	680
EATING BERRIES	0.0582	69000	860	860
EATING VEGETS.	0.1210	33000	410	410
EATING DEER	0.0131	310000	3800	3800
EATING BIRD	0.0827	48000	600	600
EATING FISH	0.0293	140000	1700	1700
<u>For Combined Routes of Exposure:</u>				
HIKER	0.2849	14000	180	180
BERRY PICKER	0.8777	4600	57	57
HUNTER	0.3807	11000	130	130
FISHERMAN	0.3142	13000	160	160
RESIDENT	0.4059	9900	120	120

^aThe plus sign (+) means "greater than."

Table C-131

Margins of Safety for Exposed Members of the Public
Accidental-Worst Case Spraying with Atrazine

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (1869.0)	SYSTEMIC NOEL (3.70)	REPRODUCTIVE NOEL (100.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.3545	5300	10	280
VEGETATION CONTACT				
HIKER	0.0051	370000	730	20000
PICKER	0.9139	2000	4.0	110
DRINKING WATER	0.1247	15000	30	800
EATING BERRIES	0.0990	19000	37	1000
EATING VEGETS.	0.2056	9100	18	490
EATING DEER	0.0222	84000	170	4500
EATING BIRD	0.1406	13000	26	710
EATING FISH	0.2494	7500	15	400
<u>For Combined Routes of Exposure</u>				
HIKER	0.4843	3900	7.6	210
BERRY PICKER	1.4922	1300	2.5	67
HUNTER	0.6471	2900	5.7	150
FISHERMAN	0.7337	2500	5.0	140
RESIDENT	0.6900	2700	5.4	140

Table C-132

Margins of Safety for Exposed Members of the Public
Accidental-Worst Case Spraying with Bromacil

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE	LD50	SYSTEMIC	REPRODUCTIVE
	(MG/KG/DAY)	(3998.0)	NOEL (6.25)	NOEL (7.92)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.4171	9600	15	19
VEGETATION CONTACT				
HIKER	0.0060	670000	1000	1300
PICKER	1.0752	3700	5.8	7.4
DRINKING WATER	0.1467	27000	43	54
EATING BERRIES	0.1165	34000	54	68
EATING VEGETS.	0.2419	17000	26	33
EATING DEER	0.0261	150000	240	300
EATING BIRD	0.1654	24000	38	48
EATING FISH	0.0587	68000	110	130
<u>For Combined Routes of Exposure:</u>				
HIKER	0.5698	7000	11	14
BERRY PICKER	1.7555	2300	3.6	4.5
HUNTER	0.7613	5300	8.2	10
FISHERMAN	0.6285	6400	9.9	13
RESIDENT	0.8117	4900	7.7	9.8

Table C-133

Margins of Safety for Exposed Members of the Public
Accidental-Worst Case Spraying with 2,4-D

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (375.0)	SYSTEMIC NOEL (1.00)	REPRODUCTIVE NOEL (5.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.1026	3700	9.7	48
VEGETATION CONTACT				
HIKER	0.0015	250000	680	3400
PICKER	0.2645	1400	3.8	19
DRINKING WATER	0.0602	6200	17	84
EATING BERRIES	0.0478	7900	21	100
EATING VEGETS.	0.0992	3800	10	50
EATING DEER	0.0103	37000	97	480
EATING BIRD	0.0645	5800	16	78
EATING FISH	0.0241	16000	42	200
<u>For Combined Routes of Exposure</u>				
HIKER	0.1642	2300	6.1	30
BERRY PICKER	0.4750	790	2.1	11
HUNTER	0.2390	1600	4.2	20
FISHERMAN	0.1883	2000	5.3	26
RESIDENT	0.2634	1400	3.8	19

Table C-134

Margins of Safety for Exposed Members of the Public
Accidental-Worst Case Spraying with 2,4-DP

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (532.0)	SYSTEMIC NOEL (5.00)	REPRODUCTIVE NOEL (6.25)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.1335	4000	37	47
VEGETATION CONTACT				
HIKER	0.0019	280000	2600	3300
PICKER	0.3441	1500	15	18
DRINKING WATER	0.0734	7300	68	85
EATING BERRIES	0.0582	9100	86	110
EATING VEGETS.	0.1210	4400	41	52
EATING DEER	0.0126	42000	400	500
EATING BIRD	0.0790	6700	63	79
EATING FISH	0.0293	18000	170	210
<u>For Combined Routes of Exposure:</u>				
HIKER	0.2087	2500	24	30
BERRY PICKER	0.6091	870	8.2	10
HUNTER	0.3003	1800	17	21
FISHERMAN	0.2381	2200	21	26
RESIDENT	0.3297	1600	15	19

Table C-135

Margins of Safety for Exposed Members of the Public
Accidental-Worst Case Spraying with Dalapon

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (7577.0)	SYSTEMIC NOEL (15.00)	REPRODUCTIVE NOEL (300.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.4171	18000	36	720
VEGETATION CONTACT				
HIKER	0.0060	1000000+	2500	50000
PICKER	1.0752	7000	14	280
DRINKING WATER	0.1467	52000	100	2000
EATING BERRIES	0.1165	65000	130	2600
EATING VEGETS.	0.2419	31000	62	1200
EATING DEER	0.0261	290000	570	11000
EATING BIRD	0.1654	46000	91	1800
EATING FISH	0.0587	130000	260	5100
<u>For Combined Routes of Exposure</u>				
HIKER	0.5698	13000	26	530
BERRY PICKER	1.7555	4300	8.5	170
HUNTER	0.7613	10000	20	390
FISHERMAN	0.6285	12000	24	480
RESIDENT	0.8117	9300	18	370

^aThe plus sign (+) means "greater than."

Table C-136

Margins of Safety for Exposed Members of the Public
Accidental-Worst Case Spraying with Dicamba

MARGIN OF SAFETY RELATIVE TO				
EXPOSURE (MG/KG/DAY)	LD50 (757.0)	SYSTEMIC NOEL (25.00)	REPRODUCTIVE NOEL (2.50)	
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0834	9100	300	30
VEGETATION CONTACT				
HIKER	0.0012	630000	21000	2100
PICKER	0.2150	3500	120	12
DRINKING WATER	0.0587	13000	430	43
EATING BERRIES	0.0466	16000	540	54
EATING VEGETS.	0.0968	7800	260	26
EATING DEER	0.0099	76000	2500	250
EATING BIRD	0.0621	12000	400	40
EATING FISH	0.0235	32000	1100	110
<u>For Combined Routes of Exposure</u>				
HIKER	0.1433	5300	170	17
BERRY PICKER	0.4037	1900	62	6.2
HUNTER	0.2153	3500	120	12
FISHERMAN	0.1668	4500	150	15
RESIDENT	0.2401	3200	100	10

Table C-137

Margins of Safety for Exposed Members of the Public
Accidental-Worst Case Spraying with Diuron

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (3750.0)	SYSTEMIC NOEL (0.63)	REPRODUCTIVE NOEL (6.25)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.6673	5600	-1.1	9.4
VEGETATION CONTACT				
HIKER	0.0096	390000	65	650
PICKER	1.7203	2200	-2.8	3.6
DRINKING WATER	0.2348	16000	2.7	27
EATING BERRIES	0.1864	20000	3.4	34
EATING VEGETS.	0.3871	9700	1.6	16
EATING DEER	0.0418	90000	15	150
EATING BIRD	0.2647	14000	2.4	24
EATING FISH	1.8780	2000	-3.0	3.3
<u>For Combined Routes of Exposure</u>				
HIKER	0.9117	4100	-1.5	6.9
BERRY PICKER	2.8088	1300	-4.5	2.2
HUNTER	1.2181	3100	-1.9	5.1
FISHERMAN	2.7897	1300	-4.5	2.2
RESIDENT	1.2987	2900	-2.1	4.8

Table C-138

Margins of Safety for Exposed Members of the Public
Accidental-Worst Case Spraying with Fosamine

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (24400.0)	SYSTEMIC NOEL (25.00)	REPRODUCTIVE NOEL (500.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.5005	49000	50	1000
VEGETATION CONTACT				
HIKER	0.0072	1000000+	3500	66000
PICKER	1.2902	19000	19	390
DRINKING WATER	0.1761	140000	140	2900
EATING BERRIES	0.1398	170000	180	3600
EATING VEGETS.	0.2903	84000	86	1700
EATING DEER	0.0313	780000	800	160000
EATING BIRD	0.1985	120000	130	2500
EATING FISH	0.0704	350000	350	7300
<u>For Combined Routes of Exposure</u>				
HIKER	0.6838	36000	37	730
BERRY PICKER	2.1066	12000	12	240
HUNTER	0.9136	27000	27	550
FISHERMAN	0.7542	32000	33	660
RESIDENT	0.9741	25000	26	510

^aThe plus sign (+) means "greater than."

Table C-139

Margins of Safety for Exposed Members of the Public
Accidental-Worst Case Spraying with Glyphosate

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (4320.0)	SYSTEMIC NOEL (31.0)	REPRODUCTIVE NOEL (10.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.2085	21000	150	48
VEGETATION CONTACT				
HIKER	0.0030	1000000+	11000	3300
PICKER	0.5376	8000	58	19
DRINKING WATER	0.0734	59000	430	140
EATING BERRIES	0.0582	74000	540	170
EATING VEGETS.	0.1210	36000	260	83
EATING DEER	0.0131	330000	2400	770
EATING BIRD	0.0827	52000	370	120
EATING FISH	0.0293	150000	1000	340
<u>For Combined Routes of Exposure</u>				
HIKER	0.2849	15000	110	35
BERRY PICKER	0.8777	4900	35	11
HUNTER	0.3807	11000	82	26
FISHERMAN	0.3142	14000	99	32
RESIDENT	0.4059	11000	77	25

^aThe plus sign (+) means "greater than."

Table C-140

Margins of Safety for Exposed Members of the Public
Accidental-Worst Case Spraying with Hexazinone

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (1690.0)	SYSTEMIC NOEL (10.00)	REPRODUCTIVE NOEL (125.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.2503	6800	40	500
VEGETATION CONTACT				
HIKER	0.0036	470000	2800	35000
PICKER	0.6451	2600	16	190
DRINKING WATER	0.0880	19000	110	1400
EATING BERRIES	0.0699	24000	140	1800
EATING VEGETS.	0.1452	12000	69	860
EATING DEER	0.0157	110000	640	8000
EATING BIRD	0.0993	17000	100	1300
EATING FISH	0.0352	48000	280	3500
<u>For Combined Routes of Exposure</u>				
HIKER	0.3419	4900	29	370
BERRY PICKER	1.0533	1600	9.5	120
HUNTER	0.4568	3700	22	270
FISHERMAN	0.3771	4500	27	330
RESIDENT	0.4870	3500	21	260

Table C-141

Margins of Safety for Exposed Members of the Public
Accidental-Worst Case Spraying with Picloram

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (8200.0)	SYSTEMIC NOEL (7.00)	REPRODUCTIVE NOEL (50.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.0100	820000	700	5000
VEGETATION CONTACT				
HIKER	0.0001	1000000+	49000	350000
PICKER	0.0258	320000	270	1900
DRINKING WATER	0.0734	110000	95	680
EATING BERRIES	0.0582	140000	120	860
EATING VEGETS.	0.1210	68000	58	410
EATING DEER	0.0118	700000	590	4200
EATING BIRD	0.0729	110000	96	690
EATING FISH	0.0293	280000	240	1700
<u>For Combined Routes of Exposure:</u>				
HIKER	0.0835	98000	84	600
BERRY PICKER	0.1674	49000	42	300
HUNTER	0.1682	49000	42	300
FISHERMAN	0.1129	73000	62	440
RESIDENT	0.2045	40000	34	240

^aThe plus sign (+) means "greater than."

Table C-142

Margins of Safety for Exposed Members of the Public
Accidental-Worst Case Spraying with Simazine

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE	LD50	SYSTEMIC	REPRODUCTIVE
	(MG/KG/DAY)	(5000.0)	NOEL (5.00)	NOEL (5.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.2085	24000	24	24
VEGETATION CONTACT				
HIKER	0.0030	1000000+	1700	1700
PICKER	0.5376	9300	9.3	9.3
DRINKING WATER	0.0734	68000	68	68
EATING BERRIES	0.0582	86000	86	86
EATING VEGETS.	0.1210	41000	41	41
EATING DEER	0.0131	380000	380	380
EATING BIRD	0.0827	60000	60	60
EATING FISH	0.0293	170000	170	170
<u>For Combined Routes of Exposure</u>				
HIKER	0.2849	18000	18	18
BERRY PICKER	0.8777	5700	5.7	5.7
HUNTER	0.3807	13000	13	13
FISHERMAN	0.3142	16000	16	16
RESIDENT	0.4059	12000	12	12

^aThe plus sign (+) means "greater than."

Table C-143

Margins of Safety for Exposed Members of the Public
Accidental-Worst Case Spraying with Tebuthiuron

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (644.0)	SYSTEMIC NOEL (12.50)	REPRODUCTIVE NOEL (90.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.2503	2600	50	360
VEGETATION CONTACT				
HIKER	0.0036	180000	3500	25000
PICKER	0.6451	1000	19	140
DRINKING WATER	0.0880	7300	140	1000
EATING BERRIES	0.0699	9200	180	1300
EATING VEGETS.	0.1452	4400	86	620
EATING DEER	0.0157	41000	800	5700
EATING BIRD	0.0993	6500	130	910
EATING FISH	0.3521	1800	35	260
<u>For Combined Routes of Exposure</u>				
HIKER	0.3419	1900	37	260
BERRY PICKER	1.0533	610	12	85
HUNTER	0.4568	1400	27	200
FISHERMAN	0.6940	930	18	130
RESIDENT	0.4870	1300	26	180

Table C-144

Margins of Safety for Exposed Members of the Public
Accidental-Worst Case Spraying with Triclopyr

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (630.0)	SYSTEMIC NOEL (2.50)	REPRODUCTIVE NOEL (10.00)
<u>For Single Route of Exposure</u>				
DERMAL, SPRAY	0.3337	1900	7.5	30
VEGETATION CONTACT				
HIKER	0.0048	130000	520	2100
PICKER	0.8602	730	2.9	12
DRINKING WATER	0.1174	5400	21	85
EATING BERRIES	0.0932	6800	27	110
EATING VEGETS.	0.1935	3300	13	52
EATING DEER	0.0209	30000	120	480
EATING BIRD	0.1323	4800	19	76
EATING FISH	0.0470	13000	53	210
<u>For Combined Routes of Exposure</u>				
HIKER	0.4558	1400	5.5	22
BERRY PICKER	1.4044	450	1.8	7.1
HUNTER	0.6091	1000	4.1	16
FISHERMAN	0.5028	1300	5.0	20
RESIDENT	0.6494	970	3.8	15

Table C-145

Margins of Safety for Doses Due to Spills of Amitrole
Accidental-Worst Case Scenario

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (4080.0)	SYSTEMIC NOEL (0.03)	REPRODUCTIVE NOEL (5.00)
<u>Spills onto Skin</u>				
CONCENTRATE	1.2000	3400	-48	4.2
SPRAY MIX	0.2400	17000	-9.6	21
<u>Spills Into Bodies of Water (Drinking One Liter of Water)</u>				
POND, HELO.	0.0737	55000	-2.9	68
RESERVOIR,HELO	0.0023	1000000+	11	2200
POND, TRUCK	1.4736	2800	-59	3.4
RESERV.,TRUCK	0.0460	89000	-1.8	110

^aThe plus sign (+) means "greater than."

Table C-146

Margins of Safety for Doses Due to Spills of Asulam
Accidental-Worst Case Scenario

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (4000.0)	SYSTEMIC NOEL (50.00)	REPRODUCTIVE NOEL (50.00)
<u>Spills onto Skin</u>				
CONCENTRATE	240.0000	17	-4.8	-4.8
SPRAY MIX	20.0400	200	2.5	2.5
<u>Spills Into Bodies of Water (Drinking One Liter of Water)</u>				
POND, HELO.	0.0615	65000	810	810
RESERVOIR,HELO	0.0019	1000000+	26000	26000
POND, TRUCK	1.2300	3300	49	41
RESERV.,TRUCK	0.0384	100000	1600	1300

^aThe plus sign (+) means "greater than."

Table C-147

Margins of Safety for Doses Due to Spills of Atrazine
Accidental-Worst Case Scenario

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (1869.0)	SYSTEMIC NOEL (3.70)	REPRODUCTIVE NOEL (100.00)
<u>Spills onto Skin</u>				
CONCENTRATE	240.0000	7.8	-65	-2.4
SPRAY MIX	24.0000	78	-6.5	4.2
<u>Spills Into Bodies of Water (Drinking One Liter of Water)</u>				
POND, HELO.	0.0737	25000	50	1400
RESERVOIR,HELO	0.0023	810000	1600	43000
POND, TRUCK	1.4730	1300	2.5	68
RESERV.,TRUCK	0.0460	41000	80	2200

Table C-148

Margins of Safety for Doses Due to Spills of Bromacil
Accidental-Worst Case Scenario

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (3998.0)	SYSTEMIC NOEL (6.25)	REPRODUCTIVE NOEL (7.92)
<u>Spills onto Skin</u>				
CONCENTRATE	240.0000	17	-38	-30
SPRAY MIX	12.0000	330	-1.9	-1.5
<u>Spills Into Bodies of Water (Drinking One Liter of Water)</u>				
POND, HELO.	---	---	---	---
RESERVOIR,HELO	---	---	---	---
POND, TRUCK	0.7365	5400	8.5	11
RESERV.,TRUCK	0.0230	170000	270	340

Table C-149

Margins of Safety for Doses Due to Spills of 2,4-D
Accidental-Worst Case Scenario

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (375.0)	SYSTEMIC NOEL (1.00)	REPRODUCTIVE NOEL (5.00)
<u>Spills onto Skin</u>				
CONCENTRATE	144.0000	2.6	-140	-1.2
SPRAY MIX	14.4000	26	-14	0.34
<u>Spills Into Bodies of Water (Drinking One Liter of Water)</u>				
POND, HELO.	0.0737	5100	14	68
RESERVOIR, HELO	0.0023	160000	430	2200
POND, TRUCK	1.4730	250	-1.5	3.4
RESERV., TRUCK	0.0460	8100	22	110

Table C-150

Margins of Safety for Doses Due to Spills of 2,4-DP
Accidental-Worst Case Scenario

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (532.0)	SYSTEMIC NOEL (5.00)	REPRODUCTIVE NOEL (6.25)
<u>Spills onto Skin</u>				
CONCENTRATE	230.4000	2.3	-46	-37
SPRAY MIX	9.6000	55	-1.9	-1.5
<u>Spills Into Bodies of Water (Drinking One Liter of Water)</u>				
POND, HELO.	0.0460	12000	110	140
RESERVOIR, HELO	0.0014	370000	3500	4300
POND, TRUCK	0.9206	580	5.4	6.8
RESERV., TRUCK	0.0288	18000	170	220

Table C-151

Margins of Safety for Doses Due to Spills of Dalapon
Accidental-Worst Case Scenario

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (7577.0)	SYSTEMIC NOEL (15.00)	REPRODUCTIVE NOEL (300.00)
<u>Spills onto Skin</u>				
CONCENTRATE	---	---	---	---
SPRAY MIX	60.0000	130	-4.0	5.0
<u>Spills Into Bodies of Water (Drinking One Liter of Water)</u>				
POND, HELO.	0.1841	41000	81	1600
RESERVOIR,HELO	0.0058	1000000+	2600	52000
POND, TRUCK	3.6825	2100	4.1	81
RESERV.,TRUCK	0.1151	66000	1.30	2600

^aThe plus sign (+) means "greater than."

Table C-152

Margins of Safety for Doses Due to Spills of Dicamba
Accidental-Worst Case Scenario

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE	LD50	SYSTEMIC	REPRODUCTIVE
	(MG/KG/DAY)	(757.0)	NOEL	NOEL
			(25.00)	(2.50)
<u>Spills onto Skin</u>				
CONCENTRATE	120.0000	6.3	-4.8	-48
SPRAY MIX	12.0000	63	2.1	-4.8
<u>Spills Into Bodies of Water (Drinking One Liter of Water)</u>				
POND, HELO.	0.0737	10000	340	34
RESERVOIR, HELO	0.0023	330000	11000	1100
POND, TRUCK	1.4730	510	17	1.7
RESERV., TRUCK	0.0460	16000	540	54

Table C-153

Margins of Safety for Doses Due to Spills of Diuron
Accidental-Worst Case Scenario

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (3750.0)	SYSTEMIC NOEL (0.63)	REPRODUCTIVE NOEL (6.25)
<u>Spills onto Skin</u>				
CONCENTRATE	240.0000	16	-380	-38
SPRAY MIX	19.2000	200	-31	-3.1
<u>Spills Into Bodies of Water (Drinking One Liter of Water)</u>				
POND, HELO.	---	---	---	---
RESERVOIR,HELO	---	---	---	---
POND, TRUCK	1.1784	3200	-1.9	5.3
RESERV.,TRUCK	0.0368	100000	17	170

Table C-154

Margins of Safety for Doses Due to Spills of Fosamine
Accidental-Worst Case Scenario

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (24400.0)	SYSTEMIC NOEL (25.00)	REPRODUCTIVE NOEL (500.00)
<u>Spills onto Skin</u>				
CONCENTRATE	240.0000	100	-9.6	2.1
SPRAY MIX	72.0000	340	-2.9	6.7
<u>Spills Into Bodies of Water (Drinking One Liter of Water)</u>				
POND, HELO.	0.2210	110000	110	2300
RESERVOIR,HELO	0.0069	1000000+	3600	73000
POND, TRUCK	4.4191	5500	5.7	110
RESERV.,TRUCK	0.1381	180000	180	3600

^aThe plus sign (+) means "greater than."

Table C-155

Margins of Safety for Doses Due to Spills of Glyphosate
Accidental-Worst Case Scenario

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (4320.0)	SYSTEMIC NOEL (31.0)	REPRODUCTIVE NOEL (10.00)
<u>Spills onto Skin</u>				
CONCENTRATE	180.0000	24	-6.2	-18
SPRAY MIX	30.0000	140	-1.0	-3.0
<u>Spills Into Bodies of Water (Drinking One Liter of Water)</u>				
POND, HELO.	0.0921	47000	340	110
RESERVOIR, HELO	0.0029	1000000+	10000	3500
POND, TRUCK	1.8413	2300	34	5.4
RESERV., TRUCK	0.0575	75000	1000	170

^aThe plus sign (+) means "greater than."

Table C-156

Margins of Safety for Doses Due to Spills of Hexazinone
Accidental-Worst Case Scenario

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (1690.0)	SYSTEMIC NOEL (10.00)	REPRODUCTIVE NOEL (125.00)
<u>Spills onto Skin</u>				
CONCENTRATE	120.0000	14	-12	1.0
SPRAY MIX	18.0000	94	-1.8	6.9
<u>Spills Into Bodies of Water (Drinking One Liter of Water)</u>				
POND, HELO.	0.0552	31000	180	2300
RESERVOIR, HELO	0.0017	980000	5800	72000
POND, TRUCK	1.1048	1500	9.1	110
RESERV., TRUCK	0.0345	49000	290	3600

Table C-157

Margins of Safety for Doses Due to Spills of Picloram
Accidental-Worst Case Scenario

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (8200.0)	SYSTEMIC NOEL (7.00)	REPRODUCTIVE NOEL (50.00)
<u>Spills onto Skin</u>				
CONCENTRATE	5.7600	1400	1.2	8.7
SPRAY MIX	1.4400	5700	4.9	35
<u>Spills Into Bodies of Water (Drinking One Liter of Water)</u>				
POND, HELO.	0.0921	89000	76	540
RESERVOIR,HELO	0.0029	1000000+	2400	17000
POND, TRUCK	1.8413	4500	3.8	27
RESERV.,TRUCK	0.0575	140000	120	870

^aThe plus sign (+) means "greater than."

Table C-158

Margins of Safety for Doses Due to Spills of Simazine
Accidental-Worst Case Scenario

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE	LD50	SYSTEMIC	REPRODUCTIVE
	(MG/KG/DAY)	(5000.0)	NOEL (5.00)	NOEL (5.00)
<u>Spills onto Skin</u>				
CONCENTRATE	240.0000	21	-48	-48
SPRAY MIX	30.0000	170	-6.0	-6.0
<u>Spills Into Bodies of Water (Drinking One Liter of Water)</u>				
POND, HELO.	0.0921	54000	54	54
RESERVOIR, HELO	0.0029	1000000+	1700	1700
POND, TRUCK	1.8413	2700	2.7	2.7
RESERV., TRUCK	0.0575	87000	87	87

^aThe plus sign (+) means "greater than."

Table C-159

Margins of Safety for Doses Due to Spills of Tebuthiuron
Accidental-Worst Case Scenario

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (644.0)	SYSTEMIC NOEL (12.50)	REPRODUCTIVE NOEL (90.00)
<u>Spills onto Skin</u>				
CONCENTRATE	---	---	---	---
SPRAY MIX	36.0000	18	-2.9	2.5
<u>Spills Into Bodies of Water (Drinking One Liter of Water)</u>				
POND, HELO.	0.1105	5800	110	810
RESERVOIR,HELO	0.0035	190000	3600	26000
POND, TRUCK	2.2095	290	5.7	41
RESERV.,TRUCK	0.0690	9300	180	1300

Table C-160

Margins of Safety for Doses Due to Spills of Triclopyr
Accidental-Worst Case Scenario

MARGIN OF SAFETY RELATIVE TO				
	EXPOSURE (MG/KG/DAY)	LD50 (630.0)	SYSTEMIC NOEL (2.50)	REPRODUCTIVE NOEL (10.00)
<u>Spills onto Skin</u>				
CONCENTRATE	240.0000	2.6	-96	-24
SPRAY MIX	48.0000	13	-19	-4.8
<u>Spills Into Bodies of Water (Drinking One Liter of Water)</u>				
POND, HELO.	0.1473	4300	17	68
RESERVOIR, HELO	0.0046	140000	540	2200
POND, TRUCK	2.9460	210	-1.2	3.4
RESERV., TRUCK	0.0921	6800	27	110

Table C-161

Lifetime Cancer Risk - Exposed Public
Small Backpack, 6.0 Acres, Routine-Realistic Scenario

EXPOSURES PER LIFETIME		2,4-D	2,4-DEP	RISK FROM EXCLUSIVE USE OF:					
				ASULAM	BROMACIL	PICLORAM	AMITROLE	GLYPHOSATE	ATRAZINE
<u>FOR A SINGLE EXPOSURE</u>									
DERMAL, SPRAY	1	1.09E-10	1.36E-09	2.10E-11	5.29E-11	4.94E-13	5.01E-10	1.61E-12	1.63E-09
VEGETATION CONTACT									
HIKER	1	1.57E-12	1.96E-11	3.02E-13	7.60E-13	7.09E-15	7.19E-12	2.31E-14	2.34E-11
PICKER	1	4.33E-10	5.40E-09	8.32E-11	2.10E-10	1.95E-12	1.98E-09	6.39E-12	6.45E-09
DRINKING WATER	1	1.70E-10	1.98E-09	1.96E-11	4.93E-11	9.59E-12	4.67E-08	1.50E-12	1.52E-09
EATING BERRIES	1	3.13E-10	3.66E-09	3.61E-11	9.10E-11	1.77E-11	8.61E-08	2.77E-12	2.80E-09
EATING VEGETS.	1	6.26E-10	7.32E-09	7.23E-11	1.82E-10	3.54E-11	1.72E-07	5.55E-12	5.60E-09
EATING DEER	1	4.01E-11	4.69E-10	4.71E-12	1.18E-11	2.20E-12	1.07E-08	3.61E-13	3.65E-10
EATING FISH	1	6.79E-11	7.94E-10	7.84E-12	1.97E-11	3.83E-12	1.87E-08	6.01E-13	3.04E-09
<u>Combined Routes of Exposure</u>									
HIKER	1	2.81E-10	3.37E-09	4.09E-11	1.03E-10	1.01E-11	4.72E-08	3.14E-12	3.17E-09
BERRY PICKER	1	1.03E-09	1.24E-08	1.60E-10	4.03E-10	2.97E-11	1.35E-07	1.23E-11	1.24E-08
HUNTER	1	4.41E-10	5.24E-09	6.01E-11	1.51E-10	1.86E-11	8.86E-08	4.61E-12	4.66E-09
FISHERMAN	1	3.49E-10	4.16E-09	4.88E-11	1.23E-10	1.39E-11	6.59E-08	3.74E-12	6.21E-09
RESIDENT	1	9.07E-10	1.07E-08	1.13E-10	2.85E-10	4.55E-11	2.19E-07	8.68E-12	8.78E-09
<u>FOR 30 EXPOSURES</u>									
DERMAL, SPRAY	30	3.28E-09	4.09E-08	6.31E-10	1.59E-09	1.48E-11	1.50E-08	4.84E-11	4.89E-08
VEGETATION CONTACT									
HIKER	30	4.71E-11	5.87E-10	9.05E-12	2.28E-11	2.13E-13	2.16E-10	6.94E-13	7.02E-10
PICKER	30	1.30E-08	1.62E-07	2.50E-09	6.29E-09	5.86E-11	5.95E-08	1.92E-10	1.94E-07
DRINKING WATER	30	5.09E-09	5.95E-08	5.88E-10	1.48E-09	2.88E-10	1.40E-06	4.51E-11	4.56E-08
EATING BERRIES	30	9.40E-09	1.10E-07	1.08E-09	2.73E-09	5.31E-10	2.58E-06	8.32E-11	8.41E-08
EATING VEGETS.	30	1.88E-08	2.20E-07	2.17E-09	5.46E-09	1.06E-09	5.17E-06	1.66E-10	1.68E-07
EATING DEER	30	1.20E-09	1.41E-08	1.41E-10	3.55E-10	6.61E-11	3.22E-07	1.08E-11	1.09E-08
EATING FISH	30	2.04E-09	2.38E-08	2.35E-10	5.92E-10	1.15E-10	5.60E-07	1.80E-11	9.11E-08
<u>Combined Routes of Exposure</u>									
HIKER	30	8.42E-09	1.01E-07	1.23E-09	3.09E-09	3.03E-10	1.42E-06	9.42E-11	9.52E-08
BERRY PICKER	30	3.08E-08	3.72E-07	4.80E-09	1.21E-08	8.92E-10	4.06E-06	3.68E-10	3.72E-07
HUNTER	30	1.32E-08	1.57E-07	1.80E-09	4.54E-09	5.59E-10	2.66E-06	1.38E-10	1.40E-07
FISHERMAN	30	1.05E-08	1.25E-07	1.46E-09	3.68E-09	4.18E-10	1.98E-06	1.12E-10	1.86E-07
RESIDENT	30	2.72E-08	3.21E-07	3.40E-09	8.55E-09	1.36E-09	6.58E-06	2.61E-10	2.63E-07

Table C-162

Lifetime Cancer Risk - Exposed Public
Large Backpack, 60 Acres, Routine-Worst Case

EXPOSURES PER LIFETIME		2,4-D	2,4-DP	RISK FROM EXCLUSIVE USE OF:			AMITROLE	GLYPHOSATE	ATRAZINE
		ASULAM	BROMACIL	PICLORAM					
FOR A SINGLE EXPOSURE									
DERMAL, SPRAY	1	4.21E-10	5.64E-09	1.13E-10	2.55E-10	3.80E-12	2.41E-09	1.03E-11	4.18E-09
VEGETATION CONTACT									
HIKER	1	6.04E-12	8.09E-11	1.62E-12	3.66E-12	5.45E-14	3.46E-11	1.48E-13	6.00E-11
PICKER	1	1.08E-09	1.45E-08	2.90E-10	6.57E-10	9.80E-12	6.21E-09	2.67E-11	1.08E-08
DRINKING WATER	1	3.91E-10	4.91E-09	6.28E-11	1.42E-10	4.42E-11	1.34E-07	5.77E-12	2.33E-09
EATING BERRIES	1	7.55E-10	9.49E-09	1.21E-10	2.74E-10	8.52E-11	2.59E-07	1.11E-11	4.50E-09
EATING VEGETS.	1	1.51E-09	1.90E-08	2.42E-10	5.48E-10	1.70E-10	5.19E-07	2.23E-11	9.00E-09
EATING DEER	1	9.73E-11	1.22E-09	1.59E-11	3.60E-11	1.07E-11	3.25E-08	1.46E-12	5.91E-10
EATING FISH	1	1.56E-10	1.97E-09	2.51E-11	5.68E-11	1.77E-11	5.38E-08	2.31E-12	4.66E-09
Combined Routes of Exposure									
HIKER	1	8.18E-10	1.06E-08	1.77E-10	4.00E-10	4.80E-11	1.37E-07	1.63E-11	6.58E-09
BERRY PICKER	1	2.65E-09	3.46E-08	5.87E-10	1.33E-09	1.43E-10	4.02E-07	5.39E-11	2.18E-08
HUNTER	1	1.21E-09	1.56E-08	2.43E-10	5.50E-10	9.02E-11	2.65E-07	2.23E-11	9.03E-09
FISHERMAN	1	9.74E-10	1.26E-08	2.02E-10	4.57E-10	6.57E-11	1.91E-07	1.86E-11	1.12E-08
RESIDENT	1	2.33E-09	2.96E-08	4.19E-10	9.49E-10	2.18E-10	6.56E-07	3.85E-11	1.56E-08
FOR 30 EXPOSURES									
DERMAL, SPRAY	30	1.26E-08	1.69E-07	3.38E-09	7.64E-10	1.14E-10	7.23E-08	3.10E-10	1.26E-07
VEGETATION CONTACT									
HIKER	30	1.81E-10	2.43E-09	4.85E-11	1.10E-10	1.64E-12	1.04E-09	4.45E-12	1.80E-09
PICKER	30	3.25E-08	4.36E-07	8.71E-09	1.97E-08	2.94E-10	1.86E-07	8.00E-10	3.24E-07
DRINKING WATER	30	1.17E-08	1.47E-07	1.88E-09	4.26E-09	1.32E-09	4.03E-06	1.73E-10	7.00E-08
EATING BERRIES	30	2.26E-08	2.85E-07	3.64E-09	8.22E-09	2.56E-09	7.78E-06	3.34E-10	1.35E-07
EATING VEGETS.	30	4.53E-08	5.69E-07	7.27E-09	1.64E-08	5.11E-09	1.56E-05	6.68E-10	2.70E-07
EATING DEER	30	2.92E-09	3.67E-08	4.77E-10	1.08E-09	3.21E-10	9.75E-07	4.39E-11	1.77E-08
EATING FISH	30	4.69E-09	5.90E-08	7.54E-10	1.70E-09	5.30E-10	1.61E-06	6.92E-11	1.40E-07
Combined Routes of Exposure									
HIKER	30	2.45E-08	3.19E-07	5.31E-09	1.20E-08	1.44E-09	4.11E-06	4.88E-10	1.97E-07
BERRY PICKER	30	7.96E-08	1.04E-06	1.76E-08	3.98E-08	4.29E-09	1.21E-05	1.62E-09	6.54E-07
HUNTER	30	3.64E-08	4.69E-07	7.29E-09	1.65E-08	2.71E-09	7.95E-06	6.70E-10	2.71E-07
FISHERMAN	30	2.92E-08	3.78E-07	6.07E-09	1.37E-08	1.97E-09	5.72E-06	5.57E-10	2.37E-07
RESIDENT	30	6.98E-08	8.88E-07	1.26E-08	2.85E-08	6.55E-09	1.97E-05	1.16E-09	4.67E-07

Table C-163

Lifetime Cancer Risk - Exposed Public
Small Right of Way, Routine-Realistic Scenario

EXPOSURES PER		RISK FROM EXCLUSIVE USE OF:					AMITROLE	GLYPHOSATE	ATRAZINE
LIFETIME	2,4-D	2,4-DP	ASULAM	BROMACIL	PICLORAM				
FOR A SINGLE EXPOSURE									
DERMAL, SPRAY	1	2.65E-11	3.30E-10	8.15E-12	1.03E-11	9.57E-14	9.71E-11	4.17E-13	3.16E-10
VEGETATION CONTACT									
HIKER	1	3.80E-13	4.74E-12	1.17E-13	1.47E-13	1.37E-15	1.39E-12	5.98E-15	4.53E-12
PICKER	1	4.43E-10	1.79E-09	4.41E-11	5.55E-11	5.18E-13	5.25E-10	2.25E-12	1.71E-09
DRINKING WATER	1	9.52E-11	1.11E-09	1.76E-11	2.21E-11	4.30E-12	2.09E-08	8.99E-13	6.81E-10
EATING BERRIES	1	1.31E-10	1.53E-09	2.42E-11	3.04E-11	5.91E-12	2.88E-08	1.24E-12	9.36E-10
EATING VEGETS.	1	2.62E-10	3.06E-09	4.83E-11	6.08E-11	1.18E-11	5.76E-08	2.47E-12	1.87E-09
EATING DEER	1	1.61E-11	1.88E-10	3.01E-12	3.79E-12	7.10E-13	3.45E-09	1.54E-13	1.17E-10
EATING FISH	1	3.81E-11	4.45E-10	7.03E-12	8.85E-12	1.72E-12	8.38E-09	3.60E-13	1.36E-09
Combined Routes of Exposure									
HIKER	1	1.22E-10	1.45E-09	2.58E-11	3.25E-11	4.40E-12	2.10E-08	1.32E-12	1.00E-09
BERRY PICKER	1	3.96E-10	4.76E-09	9.40E-11	1.18E-10	1.08E-11	5.03E-08	4.81E-12	3.64E-09
HUNTER	1	1.80E-10	2.12E-09	3.68E-11	4.64E-11	6.87E-12	3.30E-08	1.88E-12	1.43E-09
FISHERMAN	1	1.60E-10	1.89E-09	3.29E-11	4.14E-11	6.12E-12	2.94E-08	1.68E-12	2.36E-09
RESIDENT	1	3.84E-10	4.51E-09	7.41E-11	9.34E-11	1.62E-11	7.86E-08	3.79E-12	2.87E-09
FOR 30 EXPOSURES									
DERMAL, SPRAY	30	7.94E-10	9.90E-09	2.44E-10	3.08E-10	2.87E-12	2.91E-09	1.25E-11	9.47E-09
VEGETATION CONTACT									
HIKER	30	1.14E-11	1.42E-10	3.51E-12	4.41E-12	4.12E-14	4.18E-11	1.79E-13	1.36E-10
PICKER	30	4.30E-09	5.36E-08	1.32E-09	1.67E-09	1.55E-11	1.58E-08	6.76E-11	5.13E-08
DRINKING WATER	30	2.86E-09	3.34E-08	5.27E-10	6.64E-10	1.29E-10	6.28E-07	2.70E-11	2.04E-08
EATING BERRIES	30	3.92E-09	4.59E-08	7.25E-10	9.12E-10	1.77E-10	8.63E-07	3.71E-11	2.81E-08
EATING VEGETS.	30	7.85E-09	9.18E-08	1.45E-09	1.82E-09	3.55E-10	1.73E-06	7.41E-11	5.62E-08
EATING DEER	30	4.82E-10	5.64E-09	9.03E-11	1.14E-10	2.13E-11	1.04E-07	4.62E-12	3.50E-09
EATING FISH	30	1.14E-09	1.34E-08	2.11E-10	2.66E-10	5.16E-11	2.51E-07	1.08E-11	4.09E-08
Combined Routes of Exposure									
HIKER	30	3.66E-09	4.34E-08	7.75E-10	9.76E-10	1.32E-10	6.31E-07	3.96E-11	3.01E-08
BERRY PICKER	30	1.19E-08	1.43E-07	2.82E-09	3.55E-09	3.25E-10	1.51E-06	1.44E-10	1.09E-07
HUNTER	30	5.39E-09	6.37E-08	1.11E-09	1.39E-09	2.06E-10	9.91E-07	5.65E-11	4.29E-08
FISHERMAN	30	4.80E-09	5.68E-08	9.86E-10	1.24E-09	1.84E-10	8.83E-07	5.04E-11	7.09E-08
RESIDENT	30	1.15E-08	1.35E-07	2.22E-09	2.80E-09	4.87E-10	2.36E-06	1.14E-10	8.62E-08

Table C-164

Lifetime Cancer Risk - Exposed Public
Large Right of Way, Routine-Worst Case

EXPOSURES PER LIFETIME	2,4-D	2,4-DP	RISK FROM EXCLUSIVE USE OF:			AMITROLE	GLYPHOSATE	ATRAZINE	
			ASULAM	BROMACIL	PICLORAM				
FOR A SINGLE EXPOSURE									
DERMAL, SPRAY	1	1.51E-10	2.30E-09	5.90E-11	8.92E-11	6.65E-13	1.35E-09	3.62E-12	3.11E-09
VEGETATION CONTACT									
HIKER	1	2.17E-12	3.29E-11	8.47E-13	1.28E-12	9.55E-15	1.94E-11	5.20E-14	4.46E-11
PICKER	1	3.89E-10	5.92E-09	1.52E-10	2.30E-10	1.72E-12	3.48E-09	9.34E-12	8.02E-09
DRINKING WATER	1	2.09E-10	2.98E-09	4.90E-11	7.40E-11	1.15E-11	1.12E-07	3.01E-12	2.58E-09
EATING BERRIES	1	3.25E-10	4.64E-09	7.63E-11	1.15E-10	1.79E-11	1.75E-07	4.68E-12	4.02E-09
EATING VEGETS.	1	6.51E-10	9.28E-09	1.53E-10	2.31E-10	3.58E-11	3.49E-07	9.37E-12	8.05E-09
EATING DEER	1	4.05E-11	5.78E-10	9.65E-12	1.46E-11	2.18E-12	2.12E-08	5.92E-13	5.09E-10
EATING FISH	1	8.36E-11	1.19E-09	1.96E-11	2.96E-11	4.60E-12	4.48E-08	1.20E-12	5.17E-09
Combined Routes of Exposure									
HIKER	1	3.62E-10	5.31E-09	1.09E-10	1.64E-10	1.22E-11	1.13E-07	6.68E-12	5.74E-09
BERRY PICKER	1	1.07E-09	1.58E-08	3.36E-10	5.08E-10	3.18E-11	2.91E-07	2.06E-11	1.77E-08
HUNTER	1	5.13E-10	7.47E-09	1.46E-10	2.20E-10	2.01E-11	1.90E-07	8.94E-12	7.68E-09
FISHERMAN	1	4.46E-10	6.50E-09	1.28E-10	1.94E-10	1.68E-11	1.58E-07	7.88E-12	1.09E-08
RESIDENT	1	1.01E-09	1.46E-08	2.61E-10	3.95E-10	4.80E-11	4.63E-07	1.60E-11	1.38E-08
FOR 30 EXPOSURES									
DERMAL, SPRAY	30	4.53E-09	6.89E-08	1.77E-09	2.68E-09	2.00E-11	4.05E-08	1.09E-10	9.33E-08
VEGETATION CONTACT									
HIKER	30	6.50E-11	9.88E-10	2.54E-11	3.84E-11	2.86E-13	5.81E-10	1.56E-12	1.34E-09
PICKER	30	1.17E-08	1.78E-07	4.56E-09	6.90E-09	5.15E-11	1.04E-07	2.80E-10	2.41E-07
DRINKING WATER	30	6.27E-09	8.94E-08	1.47E-09	2.22E-09	3.45E-10	3.36E-06	9.02E-11	7.75E-08
EATING BERRIES	30	9.76E-09	1.39E-07	2.29E-09	3.46E-09	5.38E-10	5.24E-06	1.40E-10	1.21E-07
EATING VEGETS.	30	1.95E-08	2.78E-07	4.58E-09	6.92E-09	1.08E-09	1.05E-05	2.81E-10	2.41E-07
EATING DEER	30	1.22E-09	1.74E-08	2.90E-10	4.38E-10	6.55E-11	6.36E-07	1.78E-11	1.53E-08
EATING FISH	30	2.51E-09	3.57E-08	5.88E-10	8.88E-10	1.38E-10	1.35E-06	3.61E-11	1.55E-07
Combined Routes of Exposure									
HIKER	30	1.09E-08	1.59E-07	3.27E-09	4.93E-09	3.66E-10	3.40E-06	2.00E-10	1.72E-07
BERRY PICKER	30	3.22E-08	4.75E-07	1.01E-08	1.53E-08	9.54E-10	8.74E-06	6.19E-10	5.32E-07
HUNTER	30	1.54E-08	2.24E-07	4.37E-09	6.60E-09	6.03E-10	5.70E-06	2.68E-10	2.30E-07
FISHERMAN	30	1.34E-08	1.95E-07	3.85E-09	5.82E-09	5.04E-10	4.75E-06	2.37E-10	3.27E-07
RESIDENT	30	3.04E-08	4.38E-07	7.84E-09	1.19E-08	1.44E-09	1.39E-05	4.81E-10	4.14E-07

Table C-165

Lifetime Cancer Risk - Exposed Public
Accidental-Worst Case Spraying

EXPOSURES PER		RISK FROM EXCLUSIVE USE OF:					AMITROLE	GLYPHOSATE	ATRAZINE
LIFETIME	2,4-D	2,4-DP	ASULAM	BROMACIL	PICLORAM				
FOR A SINGLE EXPOSURE									
DERMAL, SPRAY	1	1.05E-07	1.60E-06	4.11E-08	6.20E-08	1.16E-09	9.39E-07	2.52E-09	2.16E-06
VEGETATION CONTACT									
HIKER	1	1.51E-09	2.29E-08	5.89E-10	8.90E-10	1.66E-11	1.35E-08	3.61E-11	3.11E-08
PICKER	1	2.71E-07	4.12E-06	1.06E-07	1.60E-07	2.98E-09	2.42E-06	6.50E-09	5.58E-06
DRINKING WATER	1	6.16E-08	8.78E-07	1.44E-08	2.18E-08	8.48E-09	3.30E-05	8.86E-10	7.61E-07
EATING BERRIES	1	4.89E-08	6.97E-07	1.15E-08	1.73E-08	6.73E-09	2.62E-05	7.04E-10	6.05E-07
EATING VEGETS.	1	1.02E-07	1.45E-06	2.38E-08	3.60E-08	1.40E-08	5.45E-05	1.46E-09	1.26E-06
EATING DEER	1	1.05E-08	1.50E-07	2.57E-09	3.88E-09	1.36E-09	5.28E-06	1.58E-10	1.36E-07
EATING FISH	1	2.46E-08	3.51E-07	5.78E-09	8.73E-09	3.39E-09	1.32E-05	3.55E-10	1.52E-06
Combined Routes of Exposure									
HIKER	1	1.68E-07	2.50E-06	5.61E-08	8.47E-08	9.65E-09	3.40E-05	3.44E-09	2.96E-06
BERRY PICKER	1	4.86E-07	7.29E-06	1.73E-07	2.61E-07	1.94E-08	6.26E-05	1.06E-08	9.11E-06
HUNTER	1	2.45E-07	3.59E-06	7.49E-08	1.13E-07	1.94E-08	7.19E-05	4.60E-09	3.95E-06
FISHERMAN	1	1.93E-07	2.85E-06	6.19E-08	9.35E-08	1.30E-08	4.72E-05	3.80E-09	4.48E-06
RESIDENT	1	2.70E-07	3.95E-06	7.99E-08	1.21E-07	2.36E-08	8.85E-05	4.90E-09	4.21E-06
FOR 30 EXPOSURES									
DERMAL, SPRAY	30	3.15E-06	4.79E-05	1.23E-06	1.86E-06	3.47E-08	2.82E-05	7.56E-08	6.49E-05
VEGETATION CONTACT									
HIKER	30	4.52E-08	6.87E-07	1.77E-08	2.67E-08	4.98E-10	4.04E-07	1.08E-09	9.32E-07
PICKER	30	8.12E-06	1.24E-04	3.18E-06	4.80E-06	8.95E-08	7.26E-05	1.95E-07	1.67E-04
DRINKING WATER	30	1.85E-06	2.63E-05	4.33E-07	6.55E-07	2.54E-07	9.91E-04	2.66E-08	2.28E-05
EATING BERRIES	30	1.47E-06	2.09E-05	3.44E-07	5.20E-07	2.02E-07	7.87E-04	2.11E-08	1.81E-05
EATING VEGETS.	30	3.05E-06	4.34E-05	7.14E-07	1.08E-06	4.19E-07	1.63E-03	4.38E-08	3.77E-05
EATING DEER	30	3.15E-07	4.51E-06	7.71E-08	1.16E-07	4.08E-08	1.58E-04	4.73E-09	4.07E-06
EATING FISH	30	7.39E-07	1.05E-05	1.73E-07	2.62E-07	1.02E-07	3.96E-04	1.06E-08	4.57E-05
Combined Routes of Exposure									
HIKER	30	5.04E-06	7.49E-05	1.68E-06	2.54E-06	2.90E-07	1.02E-03	1.03E-07	8.87E-05
BERRY PICKER	30	1.46E-05	2.19E-04	5.18E-06	7.83E-06	5.81E-07	1.88E-03	3.18E-07	2.73E-04
HUNTER	30	7.34E-06	1.08E-04	2.25E-06	3.40E-06	5.83E-07	2.16E-03	1.38E-07	1.19E-04
FISHERMAN	30	5.78E-06	8.55E-05	1.86E-06	2.80E-06	3.91E-07	1.42E-03	1.14E-07	1.34E-04
RESIDENT	30	8.09E-06	1.18E-04	2.40E-06	3.62E-06	7.09E-07	2.65E-03	1.47E-07	1.26E-04

Table C-166

Lifetime Cancer Risk - Exposure Due to Spills

	EXPOSURES PER LIFETIME	RISK FROM EXCLUSIVE USE OF:						AMITROLE	GLYPHOSATE	
		2,4-D	2,4-DP	ASULAM	BROMACIL	PICLORAM				
<u>FOR A SINGLE EXPOSURE</u>										
<u>SPILLS ONTO SKIN</u>										
CONCENTRATE	1	1.47E-04	2.75E-03	4.72E-05	3.57E-05	6.66E-07	3.38E-04	2.17E-06		
SPRAY MIX	1	1.47E-05	1.15E-04	3.95E-06	1.78E-06	1.66E-07	6.76E-05	3.62E-07		
<u>SPILLS INTO WATER (1 LITER DRUNK)</u>										
POND, HELO.	1	7.54E-08	5.51E-07	1.21E-08	----	1.06E-08	2.07E-05	1.11E-09		
RESERV., HELO.	1	2.36E-09	1.72E-08	3.78E-10	----	3.33E-10	6.48E-07	3.48E-11		
POND, TRUCK	1	1.51E-06	1.10E-05	2.42E-07	1.10E-07	2.13E-07	4.15E-04	2.22E-08		
RESERV., TRUCK	1	4.71E-08	3.44E-07	7.56E-09	3.42E-09	6.65E-09	1.30E-05	6.95E-10		

Appendix D
Human Health Risk
Assessment
(Quantitative)

Index

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Appendix E

Silviculture Program Effects

E

Appendix E

Silviculture Program Effects

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Program Size

The following tables describe program size by alternative and method.

Table E-1

Site Preparation Alternative

Thousand Ac. (%)

<i>Method</i>	<i>A</i>	<i>B</i>	<i>C</i>	<i>D</i>	<i>E</i>	<i>F</i>	<i>G</i>
No Treatment	10 (18)	8 (14)	0	17 (31)	8 (15)	8 (15)	6 (10)
Manual	6 (11)	3 (5)	0	3 (5)	5 (9)	9 (16)	4 (6)
Chemical	0 (0)	8 (15)	0	8 (15)	5 (9)	10 (18)	12 (19)
Mechanical	20 (36)	18 (33)	0	10 (18)	22 (40)	26 (47)	18 (29)
Biological	2 (4)	1 (2)	0	2 (4)	1 (2)	2 (4)	1 (2)
Thermal	17 (31)	17 (31)	0	15 (27)	8 (15)	0 (0)	21 (34)
Total	55	55	0	55	55	55	62

Table E-2

Conifer Release Alternative

Thousand Ac. (%)

<i>Method</i>	<i>A</i>	<i>B</i>	<i>C</i>	<i>D</i>	<i>E</i>	<i>F</i>	<i>G</i>
No Treatment	6 (17)	3 (8)	0	9 (25)	4 (11)	3 (8)	5 (10)
Manual	22 (61)	8 (22)	0	15 (42)	10 (28)	8 (22)	9 (18)
Chemical	0 (0)	21 (58)	0	7 (19)	18 (50)	21 (58)	30 (60)
Mechanical	3 (8)	1 (3)	0	2 (5)	2 (5)	2 (6)	2 (4)
Biological	4 (11)	2 (6)	0	2 (6)	1 (3)	1 (3)	3 (6)
Thermal	11 (3)	1 (3)	0	1 (3)	1 (3)	1 (3)	1 (2)
Total	36	36	0	36	36	36	50

Program Effects

There are several program effects (consequences) that apply to all alternatives. These include method effectiveness, the substitutability of methods, and for all alternatives except A, the efficacy of herbicides.

Method Effectiveness

Site preparation or release objectives defined in a site-specific silvicultural prescription can be met effectively by any method when applied to a specific performance level in the proper set of circumstances.

These site-specific factors may include limitations and opportunities within an operational environment, the defined management objectives and standards, vegetative composition and predicted response, economic constraints, and logistical considerations. Logistical considerations may include things such as the availability of equipment or labor sources, size or scale of operation constraints, seasonal “windows” of opportunity which can limit project size, the spatial distribution of treatment areas, or variables in the sequence of events needed to successfully reforest a site (such as seedling availability and quality, planting quality, etc.). Method selection—or, in many cases, a decision to defer action—can only be addressed in a site-specific diagnosis.

A moderate level of damage to crop trees during vegetation management is anticipated and considered acceptable on operational projects. This may include scattered foliar, bud or leader damage during herbicide application; seedling trampling or browsing by livestock; or the physical crushing and deformity caused by manually treated brush can become a significant site-specific factor. Examples of unacceptable herbicide damage to non-target vegetation is documented by both Newton (1978) and Gratkowski and Lauterbach (1974); while heavy damage during manual cutting is seen in work by Hobbs and Wearstler (1985) and Roberts (1980). In the majority of treatments, however, crop tree damage is either transient in nature, or the tree mortality not severe enough to cause a falldown in merchantable yields. Many of the studies used in Appendix A (Timber Growth and Yield Analysis), in fact, record an inconsistent height growth response following treatment for conifer release. This is partly a reflection of damage to individual trees. On a programmatic basis, a practice that consistently results in high levels of damage or deformity will simply be discontinued or modified to correct the problem.

Examples of successful use (i.e., meeting prescription objectives for tree establishment and early growth) of methods within these operational and biological limitations include:

Mechanical

Site preparation through machine piling, scarification, chopping, mowing, discing or ripping, crushing, scalping or terracing, has been used in relatively gentle terrain—slopes of less than 25 to 35 percent. These opportunities occur on all Forests, although most often on East-side or Transition Forests. Mechanical treatments have had limited application in the conifer release program due to the potential for excessive damage to growing stock trees. However, mowing equipment, such as the Track-Mac and Hydroaxe, have been used in some East-side or Transition Forest settings. Machine site preparation can be

especially effective where competing plants are dense and continuous (for example, many well established grass-forb complexes), or when root crowns or burls of sprouting woody shrubs must be removed. For machine treatment of sprouting species, timing can be important in order to reduce the vigor of vegetative sprouting. Operator proficiency and project administration are keys for successful manual treatments. Soil properties must be critically examined prior to use of crawler- or rubber-tired mechanical equipment for site preparation. Considerations may include soil depth and amount of coarse materials, compactability, erosion potential, nutrient distribution and mixing of soil strata, or physical obstructions. Mechanical treatments often expose mineral soils and may encourage the rapid establishment of light-seeded annual grasses and forbs, or species such as alder which are prolific seed producers.

Mechanical and thermal treatments are often used in combination during site preparation. Machine piling of logging residues and fuels is generally followed by the burning of concentrations. In other cases, machine crushing or chaining may be followed with broadcast burning.

An average cost for recent mechanical site preparation has been \$135 per acre. This will vary greatly according to circumstances and types of equipment. Some representative costs have included:

Dozer with blade	–	\$80-\$100/ac.
Ripping and piling	–	\$75-\$160/ac.
Disking	–	\$70-\$80/ac.
Track-Mac	–	\$150-\$250/ac.

Manual

Hand cutting, falling, girdling, grubbing, pulling, scalping, and mulching have been used for both release and site preparation. Scalping or mulching to reduce moisture stress to seedlings from grasses and herbaceous vegetation in new plantations is a common treatment throughout the Region. Cutting of woody shrubs or weed trees for site preparation is employed in the Coastal and southern and central Cascades subregion; often in combination with a direct herbicide application for inhibition of vegetative sprouting. All manual methods have been used for conifer release. Hand cutting, falling, or girdling have been utilized most heavily in the Coastal, central and southern Cascades, and Transition subregions. The most rapid increase in manual release has occurred on Forests which have had the largest historic herbicide use programs, within the Southern Cascades and Southern Coastal subregions. The operational effectiveness of these hand treatments is improving as knowledge is gained to better define phenological characteristics, treatment “windows,” growth potentials,

and effects on conifer survival and growth of the principal competing shrub and tree species. The duration of effectiveness of hand cutting is a concern when dealing with sprouting vegetation. As a Regionwide average, it has taken 1.8 manual cuttings compared to 1.1 herbicide treatments to adequately meet prescription release objectives. The physical volume of vegetation to be cut, and its potential for injury to growing stock seedlings, can be a limitation in the use of hand cutting in dense, older, or well-established brush fields.

Pulling or grubbing of germinants or young shrub seedlings has been employed for plantation release in the Cascades, Coastal, and Transition subregions. As with hand cutting, the most rapid increase in use of this method has occurred on Forests which have sizable historic herbicide programs. Careful consideration of soil texture, percent of coarse fragments, soil moisture, and plant rooting depth is necessary for an effective pulling project. As a general statement, all of the manual methods can be especially effective when small or isolated areas must be treated.

An average cost for manual treatments has been \$206 per acre for site preparation and \$166 per acre for conifer release. This, of course, varies greatly by specific technique. Program size and regularity from year-to-year are particularly important in the development of a Forest-level manual program, in order to create the economy-of-scale needed for operational use. Some typical costs for the different techniques have been:

Manual cutting (alder)	– \$50-\$90/ac.
Manual cutting (tanoak)	– \$150-\$500/ac.
Mulching (paper)	– \$70-\$235/ac.
Grubbing	– \$110-\$160/ac.
Pulling	– \$40-\$200/ac.

Chemical

Herbicide application through aerial (helicopter, or fixed-wing to a lesser extent), mechanical (truck-mounted or towed sprayer), pressurized backpack ground equipment, or hand methods (injection, daubing of cut surfaces, or use of granular formulations) have been effectively used for both site preparation and release. Although herbicides have been utilized on all 19 Forests, the use has been concentrated in the southern Coastal, and central and southern Cascades subregions. Chemical use in the East-side subregion has been primarily a site preparation technique. A discussion of target vegetation and the effectiveness of individual compounds is presented in Appendix C (Herbicide Use and Efficacy). The southern Cascades is where herbicides can best be used to deal with the complex of competing vegeta-

tion, seasonal drought and precipitation patterns, and limitations due to rugged terrain—which often occur in combination.

The use of systemic, translocated herbicides can inhibit resprouting of many hardwood and shrub species, thereby improving the duration of treatment effectiveness in comparison to manual cutting (see Manual). The potential for excessive damage to nontarget trees must be recognized, particularly in helicopter application for conifer release. Timing is normally critical in that application is made after crop tree height growth has ceased and buds have hardened, but while competing vegetation is still growing. Tolerance to herbicide exposure in conifers varies by tree species, type of herbicide, microclimate or aspect change, and physiologic factors related to growth of individual trees in the general population. While significant tree mortality is uncommon, damage to foliage or buds and leader deformity is serious enough to affect height growth response in an estimated 10 to 15 percent of aerial release applications. A minor program (500 to 1,000 acres per year) of herbicide use to desiccate or kill vegetation, followed in 2 to 6 months by broadcast burning, would occur in Alternatives B and G.

Average cost for herbicide use during 1983 (taken from GAO, Dec., 1986):

Aerial	–	\$56 /ac.
Ground (backpack)	–	\$132 /ac.

Biological

The prolonged or forced grazing of cattle and sheep has proven to be effective in the release program (and for site preparation to a lesser extent) when used in the correct vegetation complex and age of plantation. As with several of the manual techniques, the use of livestock for plantation maintenance is increasing as knowledge is gained in the areas of project logistics and administrative controls. The most effective operational programs can be found in the southern East-side (use of cattle) and southern Coastal (use of sheep) subregions. Grazing for release differs from the normal grazing program in that vegetation control, rather than animal weight gain or forage utilization, is the primary objective. Livestock use can be effective when palatable or preferred species are a significant component of the vegetation mix, and an area large enough to support the herd or band is available. Sheep can greatly reduce the vigor of competing shrubs and herbaceous weeds. Cattle have been effective in grassy plantations, when moisture is not a significant limitation.

A major potential advantage of the method is cost efficiency. Annual cost for a 6-year-old sheep-grazing project on the Siuslaw

National Forest has been \$12 per acre. Project costs, however, can be extremely variable, ranging up to several hundred dollars per acre. Another potential advantage is that grazing is normally socially acceptable and can be coordinated with wildlife management objectives. On some nutrient-deficient sites, the animals can be beneficial in that they convert vegetation directly into an available source of nitrogen. Grazing can also be coordinated with existing range permits, although the long-term objective is the availability of vendors that deal primarily with release contracts. Grazing is a precise method from the standpoint of administration, timing, and scale of project.

A consistent problem in the evaluation of these projects has been the establishment of a correlation between vegetation removed and the release effect in conifers. In other words, the heavy grazing of aerial plant parts may not necessarily translate into a measurable growth response in crop trees. Most potential adverse effects are related to these logistical factors. Timing is critical—utilization of competing vegetation must be complete enough to trigger the conifer release effect, but not so complete that animals start damaging seedlings.

A competent herder or rider can minimize crop tree damage by controlling animal bedding and movement. Full-time herders, however, can be difficult to find. Pasture fencing and good road access are often required. Nearby water sources are important, particularly with sheep. In some vegetation mixtures, the unpalatable competing species may simply increase as livestock reduce or eliminate the preferred plants. Inability to browse tall brush can be a limiting factor. Successful use requires careful consideration of plant palatability, crop tree size, steepness of terrain, availability of animals and herders, and adequacy of road access.

Thermal

Controlled burning prior to reforestation is commonly done over much of the Region. Burning for site preparation often has the dual objective of the reduction of potential wildfire fuels. When done under the proper prescription, broadcast burning will create a favorable seedbed for natural regeneration, create plantable spots, or reduce the physical obstructions to reforestation.

The need for site preparation through burning is often greatest on steep terrain, where mechanical methods are impossible. This can be particularly true on some highly productive sites where the volume of competing vegetation and logging residues is large. The method is used on all Forests. As with herbicides, the relative amount of site preparation burning has been decreasing as Forests look for opportu-

nities to operate within increasingly restrictive smoke management and air quality guidelines. Forests are also beginning to manage organic material for maintenance of site nutrient capital and productivity.

The need for and effectiveness of fire is sensitive to site-specific considerations, even more than other methods. Effectiveness is determined by the proper prescription—that is, control measures, fire intensity and duration, weather and fuel moisture, the desired residual large fuels and duff cover, etc. Effectiveness is also defined by a narrow time “window” in the spring or fall, when acceptable burning conditions exist. This means burning after fuels are suitably dry, but before the risk of an escaped fire is too great. Burning requires a relatively intensive administrative effort. If improperly used, broadcast burning can adversely affect site productivity. This can be especially true on high elevation sites or soil of granitic parent material, where much of the nutrient capital is contained in organic material and surface soils. The need for coordination to mitigate soil and water affects are emphasized in Chapter IV of this EIS. Cost of site preparation burns fluctuate greatly due to factors related to terrain, weather patterns, vegetation, and project logistics. The average cost has been \$238 per acre. This has ranged from \$45 to \$600 per acre in recent years.

There is much uncertainty concerning the long-term effectiveness of burning as a site preparation tool. Improved short-term seedling growth following burning has been demonstrated in studies such as those documented by Stein (1986) and Barnett (1984). There has also, however, been speculation on potential reduction in long-term site productivity due to organic matter loss, nutrient volatilization, soil erosion, reduced mycorrhizal formation or increased soil pathogens. (Barnett 1984, Perry and Rose 1985, and Perry and Tappeiner 1986, as reported in Wagner and Radosevich 1986).

Substitutability of Methods

Substitutability of methods is another major consideration within all alternatives. This means the availability of vendors or equipment, the relative effectiveness of alternative methods, project size or other logistical constraints which can limit or define management options. In a programmatic assessment, the substitutability can only be addressed in general terms. At the Forest or District level of implementation, however, these factors are often the major consideration in program design. This is also the principal advantage of the availability of several techniques within a silvicultural program.

There are some important considerations in the operational availability of these tools:

Mechanical

Some of the mechanized equipment is highly specialized, for example, the Track-Mac, Hydroaxe, and other mowing or chopping tools. In general, however, this equipment is readily available or easily adapted from timber harvest or road construction operations.

Mechanical site preparation projects can be sensitive to project size because of move-in/move-out cost considerations. As with any technique, operator proficiency and frequent monitoring is needed. This is especially critical in mechanical treatments because of the potential for considerable site or nontarget vegetation damage occurring in a short period of time.

Worker safety is always an important consideration when dealing with power tools on steep terrain or in dense brushfields.

Chemical

Herbicide use projects, particularly aerial applications, can be constrained by project size and spatial arrangement of treatment units.

In the case of release programs, timing is critical to take advantage of differential growth patterns of target and nontarget species. This means that a great deal of activity can occur during a short timeframe during spring or fall within a subregion.

Contractor availability and coordination among units is critical in order to keep programs within reasonable size limits. A large support organization and logistical planning is necessary for aerial and large ground herbicide use projects. This involves not only overhead, air operations, support, observers, and monitors, but also support activities such as resource monitoring, medial contracts, or law enforcement when needed.

Worker safety, pesticide handling, storage, and disposal must be accomplished within rigidly defined direction.

For these reasons, pesticide use projects can require a large investment of a work unit's time and energy for a short period of time, in comparison to manual or mechanical silvicultural treatments.

Manual

The availability of contractors or willing labor supply must be carefully monitored by a Forest in designing manual site preparation and release programs. Over time, contractor availability and bid rates will improve as programs increase in size and become more consistent from year to year. In the near future, however, Forests must carefully coordinate on the magnitude of manual programs to avoid "saturation" of the available vendors and willing labor pool. This has been particularly true in the case of release through use of powersaw and brush cutters.

By nature, the work is normally physically demanding, tedious, and moderately hazardous. Southwestern Oregon and northwestern California Forests have experienced some defaulting by contractors and erratic contract bid patterns. These conditions should improve in the future, but will be significant factors in manual program planning for several years.

Biological

As with manual release and site preparation, the availability of vendors and animals within a reasonable distance of a unit often limits program size. Similarly, the situation should improve in the future as grazing for silvicultural objectives becomes more commonly employed.

This technique is being examined in research and administrative studies throughout the Western United States. In the near future, however, the number of willing operators is a limitation. The use of grazing for release is a precise technique in both timing and identification of an appropriate complex of vegetation. The treatment area must be large enough to support the herd or band. Careful administration, and often a full-time herder or rider, must be found.

Forage utilized in a release effort is generally transitory in nature. This means that coordination of a project with the normal range permits is necessary. Some negotiation on grazing period or animal numbers with permittees is generally required.

Thermal

Burning for site preparation is limited to a relatively short time period in the fall or spring when fuel moistures are proper for consumption of fuels, but the risk of unacceptable resource damage is low. This time period also coincides with the bulk of burning for fuels reduction. The amount of controlled burning to be done must be carefully matched to this "window" of opportunity.

The use of fire for silvicultural purposes often requires some previous treatment of vegetation in order to achieve objectives. Because fire or heat results in particulate matter production, units must comply with state smoke management guidelines set by agreements with individual states. This establishes limits on program size or timing.

The administrative impact of a burning program can be comparable to a large herbicide project. Personnel needs include the staffing or equipping of fire lines and control points, fire-weather support, road or traffic control, safety support, and mop-up or reconnaissance crews. Logistical factors such as these are major considerations in the

development of an operational burning program at the Forest level.

For those alternatives in which herbicide use is part of the silvicultural vegetation management program (B, D, E, F, and G), the efficacy of individual compounds has a large influence on effects or consequences.

Appendix C contains an assessment of herbicide historical use and trends, rates of application, modes of action, specific plant species controlled, and potential negative effects on nontarget plants. It also discusses some important use limitations for 16 herbicides—2,4-D, glyphosate, picloram, triclopyr, atrazine, dalapon, 2,4-DP, hexazinone, fosamine, dicamba, tebuthiuron, asulam, diuron, simazine, amitrole, and bromacil.

The analysis requirements and decision process to be followed for the potential use of any new products or expanded Environmental Protection Agency label uses are also addressed in Appendix C.

Consequences of the Silvicultural Program which are Specific to One Alternative

Appendix A contains the analysis of yield effects in the absence of vegetation management for stand establishment and early maintenance to control site competition for moisture, light, or nutrients. The reduced volume yields over time is related to factors such as:

- A delay in early stand development due to vegetative competition that results in extended rotation length. There may be either a prolonged culmination of growth, or a delay in reaching a desired product size.
- An increase in tree mortality, vigor, or form defects, and suppression of growth that results in dead or submerchantable trees within a managed stand. This effect will translate into reduced commercial thinning opportunities and understocked areas within the stand.
- Restrictions on vegetation management options which lead to changes in the timberland suitability classification being made in the Forest land management planning. Specifically, these are conditions where the unavailability of vegetation management presents a high risk of regeneration failure.
- A shift in species composition in response to early vegetative competition in mixed species stands. This effect can make factors such as growth patterns, susceptibility to physical damage or pathogens, or

Efficacy of Herbicides

Timber Growth and Yields in the Absence of Vegetation Management (Alternative C)

reduced product values a concern.

Yield effects are assessed for six vegetative complexes to present a cross-section of conditions in the Region, and to take advantage of the most pertinent available literature and information. Yield reductions are then estimated, based on a comparison to typical rotation length, site quality, and management strategy being used on intensively managed sites. These are the acres being managed for full or nearly full yields as projected in Forest Plan managed yield tables.

Vegetation Complex	Yield Reduction in the Absence of Vegetation Management (%)
Douglas-fir/Red Alder	25%
Douglas-fir-Hemlock/Salmonberry/Herbaceous	21%
Ponderosa Pine/Grasses-Herbaceous	52%
Douglas-fir-Ponderosa Pine/Ceanothus spp./Herbaceous	39%
Mixed Conifer/Tanoak-Pacific Madrone	65%
True fir-Hemlock/Shrub/Grasses-Herbaceous	56%

Timber Growth and Yields in the Absence of Herbicide Use (Alternative A)

Appendix A contains a related analysis, the yield effects in the absence of herbicide use for site preparation and release. The analysis considers biological effects, operational constraints, and physical limitations that may result in reduced treatment effectiveness in the absence of herbicide use.

Cost-of-doing-business and economic efficiency are not constrained in Appendix A. The budget levels and economic thresholds for alternatives are addressed in Chapter IV (Environmental Consequences).

Potential yield reductions are estimated for the loss of herbicide use in six vegetative conditions.

Vegetation Complex	Yield Reduction in the Absence of Herbicide Use (%)
Douglas-fir/Red Alder	(None)
Douglas-fir-Hemlock/Salmonberry/Herbaceous	(None)
Ponderosa Pine/Herbaceous	4%
Douglas-fir-Ponderosa Pine/Ceanothus spp./Herbaceous	5%
Mixed Conifer/Tanoak-Pacific Madrone	19%
True fir-Hemlock/Shrub/Herbaceous	7%

Reforestation in the Absence of Prescribed Fire For Site Preparation

The use of fire as a site preparation tool will not be available under Alternative F. The magnitude of the burning program in general has been reduced in recent years as efforts are made to meet smoke management guidelines and reduce particulate emissions. This is particularly true of Forests west of the Cascade Crest. Within a Forest vicinity or major airshed, this requires coordination with other agencies and private timber operations, and agricultural and industrial programs.

When used in the proper setting, prescribed fire will reduce physical barriers to reforestation, create plantable spots or microsite conditions, or prepare a seedbed for natural regeneration.

In certain situations, the suspension of burning will have a negative effect on reforestation success. Site preparation, through use of fire, is commonly employed on steep terrain (40-plus percent) and often on highly productive sites where the physical volumes of unwanted vegetation may be large. Opportunities for substitution of other methods in lieu of fire are discussed in the following section.

The lack of fire availability, when it is needed for reforestation site preparation, will have four negative effects.

1. A reduced suitable timberland base available for intensive management: in extreme cases, the competing vegetation will present an unacceptable risk of regeneration failure. Minimum acceptable stocking standards for newly established stands (certified as successful) are developed for combinations of timber type and site productivity. A high probability of failure to meet minimum standards, based on lost opportunities for site preparation, would mean reclassification of these sites as nonregenerable. This would mean removal of the site from the "suitable" land base in the Forest planning process.

2. An increase in nonstockable inclusions in managed timber stands: the effect is similar to the previous suitable land adjustment, but it occurs in areas too small to be identified in normal land allocation mapping and monitoring. A typical effect is the presence of concentrations of undesirable vegetation or logging residues which create physical barriers and obstructions to planting. Timber yields available from a stand must therefore be adjusted downward to accommodate the nonstocked or poorly stocked holes.

Based on Forest estimates, the combined effect (suitable land adjustment, plus inclusions) due to the lack of burning for site preparation is estimated to be 2-1/2 to 3 percent of the acres programmed for full or nearly full timber yields. The major impact would occur on Forests within the northern Coastal and southern Cascades subregions. Moderate adjustments would also occur on certain Forests in the East-side and Transition subregions.

3. A reduced tree growth and vigor in newly established stands: in some vegetative conditions, there will be an increased time lag in successful reforestation, or a reduction in early tree growth and vigor due to vegetative competition. These effects will be highly dependent on site-specific conditions such as vegetation competition and stage of development, and limitations presented by the operating environment. Negative effects will be significant on sites with dense or older pre-existing vegetation, and those in which timely reforestation efforts have not occurred.

The growth and yield effect in these situations would be comparable to the “no vegetation” management effects presented in Appendix A (Analysis of Timber Growth and Yield Effects).

The use of fire as a site preparation tool for reduction of inter-species competition must be carefully diagnosed as fit for the appropriate site conditions. If ground fire intensity and duration is not controlled, there may be adverse impacts related to site productivity and nutrient levels, as well as other soil and water considerations. Negative effects can also be related to the type, vigor, and amounts of competing vegetation if fire is used in the wrong situations. The end result will be a more difficult reforestation situation and a need for followup corrective action.

Examples may be the aggressive sprouting of woody shrubs or tree species, or the triggering of germination of seedloads stored in ground litter and surface soils. Many of the widespread species of competing vegetation maintain long-term seed viability. The use of controlled fire often involves a balancing of fire fuels reduction goals with silvicultural and reforestation needs.

4. An increased loss of standing timber inventory due to wildfire: there will be an estimated 22,000 acres per year burned under Alternative F due to the increased wildfire occurrence. This means that timber growing stock and merchantable volume will be destroyed. Many of the fires would likely initiate in stands where the treatment of logging residues and activity fuels has not occurred. This probably means that a relatively high proportion of young stands would be involved in the total burned-over area.

While difficult to quantify, the effect on the silvicultural program would probably be insignificant when viewed from a Region-wide perspective. On the Ranger District or project planning level, however, this can be a significant effect on both the reforestation and timber harvest planning programs. Both activities involve a systematic sequence of events over a several year period. Disruptions caused by events such as large fires can adversely affect both the amount and quality of program accomplishment at the District level.

The consequences of a suspension of burning for site preparation will be extremely variable on individual National Forests. A quantitative assessment of a reduced success in reforestation efforts can only be made within the context of site-specific considerations and limitations.

Mechanical Methods

Mechanical site preparation is accomplished at high levels in Alternatives A, B, F and G. Moderate levels are used in Alternatives D and E, and none in Alternative C.

The effect of high level programs will be an improved record of early stand establishment. Most of the program increase over Alternative B levels would occur on East-side and Transition subregion Forests, where terrain is more suitable for mechanized equipment. There is little difference in mechanical site preparation between Alternatives A, B, and F.

This indicates that opportunities for substitution of methods, such as thermal or chemical, for mechanical are relatively limited. The site preparation remains relatively large in Alternative D. This reflects the fact that site preparation, by any method, is preventive in nature and will reduce the need for subsequent corrective actions in young stands.

Mechanical release through the use of mowing and chopping equipment is limited in all alternatives. Effects such as the potential excess damage to crop trees and marginal treatment effectiveness make the technique of relatively limited value in many situations.

Chemical Methods

Site preparation with herbicides is accomplished at a very high level in alternative G, and large programs are maintained in Alternatives F and B.

The reduced use of chemicals in Alternative E is related to specific concerns for worker or public exposure to certain herbicide compounds. Herbicide use in Alternative D is greatly reduced. There is no use of chemicals under Alternatives A or C.

As with all methods, increased use of herbicides in the appropriate site conditions and time will improve tree numbers and vigor in newly established stands. The increased use of herbicide site preparation under Alternative G will tend to be concentrated in East-side and Transition subregion Forests.

Herbicide release follows the same pattern as site preparation under various alternatives.

There is a large reduction of release with herbicides under

Silvicultural Program Effects by Alternative

Alternative D, which is proportionately greater than the change in herbicide site preparation. This reinforces the opinion that aggressive site preparation is normally preventive in nature. Alternatives with large herbicide release programs will result in improved growth and vigor of many managed stands (See Appendix A).

Manual Methods

Site preparation through scalping, grubbing, and mulching is done at high levels under all alternatives, except C. This reflects the importance of early control of grasses and herbaceous vegetation in plantations. The proportionate increase is largest under Alternatives A, F, and D. This reflects a relative shift away from chemical or thermal use in these alternatives. The greatest relative increases in manual site preparation would occur on Forests within the East-side, Transition, and southern Cascades subregions. A general effect of large site preparation programs is increased success in planted seedling survival.

Large manual release programs will occur under Alternatives A, D, and F. More moderate sized programs would be seen under Alternatives G, B, and E. The large programs reflect additional restraints on herbicide use under Alternatives A and D. Increased manual release in Alternative F is related to the need for subsequent corrective action in the absence of thermal site preparation in some circumstances. The greatest relative increase in manual release will occur in the central and southern Cascades, and southern Coastal subregions. Large programs of manual release will have the effect of improved growth and vigor in young managed stands. The duration of effectiveness is somewhat shorter than chemical release. Region-wide it has taken 1.8 manual treatments compared to 1.1 herbicide applications to achieve prescription release objectives. The need for more than a single manual release will be most common in the southern Coastal and southern Cascades subregions.

Biological Methods

Opportunities for increased use of biological techniques are greater in the release program than in site preparation. There will be a slight increase in the use of forced grazing for site preparation under alternatives A, D, and F.

Genetic adaptation through the Regional Tree Improvement Program also has favorable effects on seedling survival and vigor. Trees have evolved heritable properties to deal with competing vegetation and site limitations. Seed and seedlings produced under a genetics improvement program may increase the effectiveness of site preparation. Several biological techniques have potential value in the

silvicultural vegetation management program, but are not yet operationally effective (see Chapter II, Program Areas).

There will be an increased use of livestock grazing for plantation release under alternatives A, D, E, and F. Alternative B would see a more modest increase in the use of grazing. In the right mix of vegetation and under strict administrative control, the use of sheep or cattle can effectively produce a conifer release effect. Increased use of cattle will occur in the East-side and Transition subregions. Release through sheep grazing will become more common in the southern Cascade and Coastal subregion. Cattle use will occur primarily in grassy plantations. Sheep grazing can effectively reduce amount and vigor of herbaceous weeds and woody shrubs, in addition to grasses.

Thermal Methods

The use of fire as a site preparation tool is greatest under Alternatives G and A. Large programs will also be seen under Alternatives B and D. The reduced use of site preparation through burning in Alternative E will slightly reduce the potential for adverse health effects due to air toxics or particulate matter. No thermal site preparation occurs under Alternatives F and C.

In many natural regeneration efforts, the use of fire is an important step in seedbed preparation. Loss of burning would reduce the effectiveness of regeneration efforts in these situations. Increases in the use of fire for site preparation would occur primarily in the Coastal and Cascades subregions, where steep terrain and high volumes of competing vegetation or logging residues make burning particularly effective. The use of fire to eliminate physical barriers and obstructions for planting is an important technique on Forests within the central and southern Cascades, and Coastal subregions.

Controlled burning has limited value in conifer release, other than in ponderosa pine stands of the East-side subregion.

Deferred or Nontreatment

This is an appropriate and common action in silvicultural diagnosis and prescription under all alternatives. Increases in the number of no-action decisions are related to the damage thresholds and acceptable levels of uncertainty established in each alternative. No-action decisions will be most common under Alternative D, in response to an emphasis on data collection and analysis, increased monitoring needs, and caution in creating situations which require followup corrective treatments. There will also be an increased willingness to delay action until research or administrative studies establish more conclusive evidence regarding species interactions, beneficial effects, and long-

term site productivity implications of vegetation control. No-action is also a major component of Alternatives A, B, E, and F. Alternative G will result in relatively aggressive management, an increased tolerance for uncertainty or missing research, and an increased willingness for corrective action in the site preparation and release programs.

A combination of the lack of herbicide use since 1983, and budget restraints has created a backlog of untreated release needs. These deferred treatment areas represent 30,000 to 40,000 acres, concentrated in the southern Cascades subregion. In certain combinations of competing vegetation, terrain, and precipitation patterns, there is an increased treatment effectiveness related to herbicide use (See Appendix A). This is primarily due to the inhibition of site reoccupancy by aggressively sprouting species. Alternatives which preclude the use of chemicals (A and C) would result in stocking or vigor loss within this backlog acreage.

Appendix F

Rangelands of the Pacific Northwest Region

F

Appendix F

Rangelands of the Pacific Northwest Region

Nearly pure stands of ponderosa pine occur throughout eastern Oregon and Washington. The structure of these stands varies, based on past management. Desirable understory grasses include Idaho fescue on central Oregon sites, pinegrass/elk sedge on northeastern Oregon and eastern Washington sites. White (grand) fir sites generally support, as desirable herbaceous understory, pinegrass or elk sedge. Major shrubs in central to southern Oregon include bitterbrush, mountain mahogany, snowbrush, manzanita, and big sagebrush. Northeastern Oregon and eastern Washington sites feature common snowberry, shiny leaf spirea, mountain snowberry, rose, and to a lesser amount, ninebark. Even in areas with a closed forest canopy, these shrubs and grasses still provide manageable and desirable forage.

The Cascade Range creates a prominent rain shadow which, in turn, greatly affects the vegetation of eastern Oregon and Washington. Vast areas of grassland and shrub/grasslands are found throughout most of eastern Oregon and central and southeastern Washington.

Grasslands

The most typical perennial bunchgrasses in this area include bluebunch wheatgrass, Idaho fescue, Sandberg's bluegrass, basin wildrye, and Thurber's needlegrass. The relative abundance of these five major grasses is variable.

The two most common plant communities are predominantly Idaho fescue with associated bluebunch wheatgrass. There are several recognized variations. The second major grouping is where bluebunch wheatgrass dominates over Sandberg's bluegrass. As was the case with Idaho fescue and bluebunch wheatgrass, even though these two bunchgrasses are the most abundant on a given site, there may be a variety of native herbaceous plants found in association with them.

These sites exhibit different productivities and reaction to management. Pure stands of bunchgrass are very productive and are generally on more moist sites than areas supporting big sagebrush and bunchgrass.

Shrub/ Bunchgrass

Sites supporting both shrubs and bunchgrasses are far more common throughout eastern Oregon and Washington than the previously described pure grasslands. Typical communities support major shrubs such as big sagebrush, antelope bitterbrush, rigid sagebrush, low sagebrush, snowberry, rose, mountain mahogany, and shadscale, in addition to the perennial bunchgrasses previously discussed (Table 4). The most common shrubland association is represented by big sagebrush/bunchgrass, and is widespread throughout eastern Oregon and Washington.

In Oregon, shrub/bunchgrass sites are generally higher in elevation than are the Washington shrublands. Deep, loamy soils are not as common in eastern Oregon. Additionally, meadow grasslands supporting sod-forming grasses and herbs, characteristic of southeastern Washington, are nearly absent in eastern Oregon.

Juniper/Shrub/ Bunchgrass

Western juniper is a fairly short (up to 40' in height) conifer widespread throughout eastern Oregon, but mostly absent in eastern Washington. The combination of juniper/shrub/bunchgrass is very common in central Oregon, but only occurs as isolated stands on rocky sites or rims in most of eastern and southeastern Oregon. Most Washington sites support little or no juniper. The abundance of juniper shrublands today is attributed to lack of naturally occurring fire. Juniper has very thin bark, and is susceptible to fires. This probably explains its association with rocky sites, as in these areas there is little ground fuel to carry a fire to the tree.

The productive capability of juniper sites is similar to that of shrub/grasslands, but juniper may greatly modify the site. There is often need to control juniper growth or expansion since the tree is a strong competitor for moisture, nutrients, and as it grows, tends to block sunlight from ground vegetation.

Meadowlands

Meadowlands are very productive sites found over a wide variety of growing conditions. They typically support sod-forming grasses and sedges. Meadowlands in good condition also support a variety of herbs which are not, however, a dominant component of the vegetation.

There are three major subdivisions applied to meadowlands: 1) dry, 2) moist, and 3) wet. These sites are productive and often near a

water source. These conditions attract livestock, and these sites rarely exhibit good native conditions.

The distinctions between dry and wet meadows may best be characterized in relation to water and vegetative composition. Most dry sites are mid-elevation and lower, and support primarily the introduced—but highly palatable—Kentucky bluegrass. These sites tend to occur on better-drained soils, and are generally the greatest distance from ground water.

Moist meadowlands are dominated by a combination of tufted hairgrass, a lesser amount of Kentucky bluegrass, and a number of sedges. Slightly closer to ground water than the dry meadowlands, these sites stay wetter for longer periods of time. More woody vegetation is also associated with the moist sites in the form of willow, alder, and aspen.

Almost imperceptibly lower in elevation, the wet meadowland type occurs either as a small narrow inclusion adjacent to intermittent or perennial streams or in large wet basins. The dominant vegetation is generally mostly short to tall coarse sedges to the almost total exclusion of grasses. Willow and alder are the major woody plants. These sites will commonly exhibit surface moisture into September.

Further upslope in the subalpine setting, a variety of other meadowland types are recognized. These sites may occur as pure

Table F-1

**Summary of Forest and Rangeland Under Other Ownership
(Thousand Acres)**

<i>State</i>	Forest land		Rangeland	
	<i>Other federal</i>	<i>Non-federal</i>	<i>Other federal</i>	<i>Non-federal</i>
Oregon	4,938.0	11,112.1	11,312.7	9,186.9
Washington	1,050.3	24,818.8	1,019.7	6,227.4
Total	5,988.3	35,930.9	12,332.4	15,414.3

Table F-2

Percent of Land Grazed Under Each Ownership

<i>State</i>	<i>Other federal</i>	<i>Non-federal</i>
Oregon	79	70
Washington	38	58

Table F-3

Summary of Range Statistics for National Forests in the Pacific Northwest: Fiscal Year 1986

	<i>Oregon</i>	<i>Washington</i>
Number of grazing allotments	599	186
Number of active grazing permits	712	200
Number of cattle authorized to graze	122,100	22,400
Animal unit months (cattle)*	546,700	113,900
Number of sheep authorized to graze	44,900	10,000
Animal unit months (sheep)*	50,800	9,700
Number of horses authorized to graze	7,462	21,100
Animal unit months (horses)*	2,300	3,700
Number of wild horse territories	2	0

*One animal unit month (AUM) is the forage requirement for one month for a 1,000 pound mature animal (cow) or its equivalent (5 sheep).

Table F-4

Summary of the Six Major Ecosystems Grazed in Eastern Oregon and Washington on National Forest Lands (1987)*

	Meadows	Grassland	Shrubland	Juniper	Timber Shrubland	Timber Transitory
<i>Okanogan</i>	12,000		62,000		414,000	228,900
<i>Wenatchee</i>	43,300	65,000	100,300		192,500	41,256
<i>Colville</i>	8,500				165,500	720,000
<i>Umatilla</i>	12,317	212,360	43,936	21,224	860,100	236,149
<i>Wallowa-Whitman</i>	7,958	501,624	22,621	4,724	807,677	809,560
<i>Malheur</i>		43,199	103,527	55,692	431,305	678,685
<i>Ochoco</i>	18,400		97,350	184,900	687,450	43,900
<i>Deschutes</i>	5,773		31,507	4,917	150,000	1,311,761
<i>Fremont</i>	37,283		138,731	171,000	238,000	111,000
<i>Winema</i>	12,294		10,643	13,507	325,090	294,762
<i>Mt. Hood</i>	2,652		159		6,636	265,769
Total	189,574	822,183	610,774	456,064	4,278,758	4,741,746

Source—Personal communication with listed forest.

*These acres represent only those acres that are considered suitable in terms of vegetation potential and steepness of slope.

meadows, shrub/meadows, or sub-alpine parklands. Dominant vegetation includes herb/sedge, heath-huckleberry/herb, or high elevation conifers extending into the aforementioned settings. On some of the drier sites, green fescue, a bunchgrass, is dominant with only limited potential for shrub growth.

The total acres considered to be transitory range in forested settings is significant (Table 4) and constitute a larger acreage than the yearly grazed forested/shrub/grass types. The transitory acres found in forestlands are generally very productive due to deep soils and increased precipitation. Although ponderosa pine may still be codominant in specific locations, there is a noticeable presence of grand fir, white fir, larch, and Douglas-fir. One or more of these species may grow in combination with ponderosa pine. Usually there is considerable diversity in shrubs after the site has been logged.

In addition to the major shrubs previously discussed, two evergreen shrubs are important. Following intensive logging activity, snowbrush, a moderately palatable low-statured shrub up to 2 meters in height, or manzanita, generally a non-palatable shrub of similar stature, will often form a continuous canopy across the treated area. While snowbrush offers some browse and manzanita little to none, both plants are aggressive seral species. When fully developed, they may essentially eliminate all herbaceous vegetation by shading and through competition for nutrients and moisture.

Transitory forested rangelands often occur at midslope and higher location on the landscape. Even though these sites are very diverse and productive the range opportunities are often limited, due to aggressive shrub development and total tree/shrub competition. This often leads to heavy shading and elimination of herbaceous vegetation. On the other hand, the high elevation stands may exhibit low plant diversity, but offer few range opportunities due to the often extreme environment.

Transitory Range

Appendix G

Resource Programs and Vegetation Management Activities

G

Appendix G

Resource Programs and Vegetation Management Activities

This appendix contains a discussion of Forest Service resource management programs that would be affected by selection of one of the vegetation management alternatives. These include plantation site preparation, conifer release, fire management activities, range improvements, noxious weed control, wildlife habitat improvement activities, maintenance of recreation facilities and administrative facilities, rights-of-way, tree genetics activities, and research.

Reforestation— Site Preparation and Release

Site preparation for establishment of artificial or natural regeneration in managed timber stands is often necessary for prompt reforestation success in the Pacific Northwest Region. It commonly involves control of competing vegetation in conifer plantations for management of several components of the seedling's environment. These can include: soil water availability, light availability for shade-intolerant species, reduction of pathogens in surface litter, limiting habitat or food sources for seedling damaging rodents and invertebrates, physical injuries from litter fall, reduction of fire fuels that hamper future stand protection, and reduction of physical barriers to tree planting.

Release is the practice of controlling the density, composition, and vigor of competing vegetation during the period of seedling establishment. The objective is to maintain satisfactory survival and early development of the desirable vegetation. The environmental

components being managed are similar to those in site preparation activities. Site preparation and release are utilized within the first five to 10 years in the life of managed tree stands, and may often be done as dual purpose projects. A common goal is to control the relative dominance of crop trees, excess hardwoods, and shrub or herbaceous vegetation while seedlings develop to a size and rooting depth which ensures their survival and future growth. A key factor in the success of release efforts in all provinces has been early treatment following site disturbance through logging or fire. Prompt treatment, when competing or invading vegetation size and density is relatively manageable, allows flexibility in method selection and a high probability of meeting prescription objectives.

**Statutory
Authority,
National Program
Goals, and
Existing Policy**

Basic authority for these silvicultural activities is contained in the following laws:

1. Organic Administration Act of 1897
2. Knutson-Vandenberg Act of 1930
3. Bankhead-Jones Farm Tenant Act of 1937
4. Granger-Thye Act of 1950
6. Supplemental National Forest Reforestation Act of 1972
7. Forest and Rangeland Renewable Resources Planning Act of 1974
8. National Forest Management Act of 1976
9. Reforestation Trust Fund, Title III, Reforestation, Recreation Boating Safety and Facilities Improvement Act of 1980

Policy Statements include six principal directives, which are further defined in Agency technical handbooks. These directives are:

1. Use only those practices best suited for the land management objectives of the area.
2. Prescribe treatments that are practical in terms of preparation and administration costs.
3. Monitor practices to determine that objectives are met.
4. Prior to scheduling stands for regeneration harvest, assure, based on literature, research, or local experience, that stands being managed for timber production can be adequately restocked within 5 years.
5. Inform minorities and women about opportunities in contracting for activities.
6. Perform all silvicultural activities in the most cost efficient manner consistent with resource management objectives.

The young stand maintenance program in the Pacific Northwest Region is a significant portion of total Forest Service accomplishment in this area. In recent years, the site preparation program has averaged 45,000 to 75,000 acres annually, while plantation release has averaged 30,000 to 55,000 acres. Yearly fluctuations will occur on a forest as a result of silvicultural systems or logging activity, budgets, seedling availability, weather or personnel limitations, priorities identified in the Forest Plan, or special programs.

All methods have proven successful in meeting site preparation and release objectives under the appropriate biological and operational conditions.

Methods

Herbicides

While herbicides have been utilized on all 19 National Forests in their reforestation programs, the technique has been concentrated on certain Forests. For example, in 1982, four Forests (Siskiyou, Siuslaw, Umpqua, and Willamette) accounted for 62 percent of the herbicide treatment acres. Herbicide need and effectiveness is greatest where a combination of the complex of competing vegetation, seasonal drought and precipitation patterns, and relatively rugged or remote terrain limitations exist in combination. These qualities exist within portions of the Siskiyou and Western Cascade Provinces.

Mechanical

The use of machine piling, mowing, disking, crushing, or terracing can be effective on relatively gentle terrain: slopes of less than 25 to 35 percent. This technique is principally used in the site preparation program. Mechanical treatment can be effective in removal of root crowns of sprouting shrubs, where excessive soil movement or compaction can be avoided. The method is most often used where physiography is less limiting, such as the Upper Basin and Range, Harney Basin, and Blue Mountain Provinces.

Manual

Hand felling girdling, grubbing, pulling, scalping, mulching, and shading have proven effective when applied in the appropriate circumstances. Manual release methods, in particular, have become increasingly important since the 1983 U.S. District Court injunction on herbicide use within the Pacific Northwest Region. Manual techniques have been most effectively used in moderately severe competing vegetation (for example, red alder or several of the ceanothus species) when costs, work force limitations, and period of treatment effective-

ness can be managed to achieve prescription objectives. Manual methods are utilized Region-wide, but Forests with historically large plantation maintenance programs have been most aggressive in their utilization.

Biological

Domestic livestock—sheep and cattle—have been the most significant biological factor in the silvicultural program. Prolonged or “forced” grazing and browsing can reduce grasses, herbaceous, and palatable shrub competition to the point that a release effect is seen in the crop trees. The animals, especially sheep, must be carefully controlled and herded to achieve the desired results. This has proven to be cost effective when factors such as band or herd availability, and a relatively large administrative impact are not limitations. Sheep use has proven effective within the Coast Range Province. The potential for cattle use is greatest in the Harney Basin and Upper Basin and Range Provinces which are characterized by pine and grass-shrub plant communities.

Burning

Broadcast burning is a common treatment prior to reforestation. While the technique is primarily used for removal of logging residues and fuel accumulations, it can often have a site preparation objective. This may involve creation of a favorable seedbed for natural regeneration, creation of planting sites, or reduction of physical obstructions to reforestation. Broadcast burning is most commonly used in clearcuts on steep slopes where machines cannot be used.

Limitations on the technique involves smoke management and air quality standards, a relatively narrow prescription window—when fires and weather conditions allow effective burning at a low risk of escape—and potential adverse soil nutrient or vegetation response effects. In some situations, a ground fire of moderate intensity may trigger germination of brush or weed seed loads, or vegetative sprouting, which can aggravate the site competition for moisture, nutrient, and light. Burning for silvicultural objectives has been effectively used over a large portion of the Region.

Combinations of Methods

Several combinations have been effectively used to meet silvicultural objectives:

- Machine piling of logging residues and fuels, followed by burning of concentrations.

- Machine crushing or chaining, followed by broadcast burning.
- Aerial herbicide use to desiccate or kill vegetation, followed in 2 to 6 months by broadcast burning.
- Hand felling of hardwoods or large woody shrubs, followed by burning.
- Hand felling and daubing of cut surfaces with systemic herbicides.
- Hand cutting of large stems and injection of a systemic herbicide for translocation to the root system and aerial plant parts.

Spatial limitations are often a factor in method preference. The number and distribution of treatment areas must be large enough to allow an administratively efficient project. These project size considerations are particularly important in grazing of livestock, burning, and aerial herbicide treatments in the site preparation or release programs.

Management intensity is also an important dimension in determination of methods. Aggressive or prompt plantation establishment and release is normally limited to lands suitable for intensive management—for timber yields approaching the biological potential on a given site. Biological potentials are defined by stocking levels, growth rates, and timber yields in development of the Forest Land Management Plans. On lands where timber yield levels are reduced or secondary in importance to other resource values, the no-treatment or deferred treatment may sometimes be the preferred silvicultural option.

Methods and techniques with limited or very site-specific application: several techniques have limited use in site preparation and conifer release projects. They include:

Biological herbicides: naturally-occurring microbial and several agents have proven effective in insect suppression and agriculture. Use in forest vegetation management is not yet operationally effective.

Fertilization: fertilizers have been used to control vegetation composition in some roadside maintenance situations, but use in plantation maintenance has not been demonstrated.

Genetic adaptation: trees have evolved heritable properties to deal with competing vegetation and site limitations. Seed and seedlings developed by the Region's Tree Improvement Program may eventually limit the need for release in certain situations.

Firewood removal: commercial or personal-use roundwood removal for fuelwood can reduce unwanted vegetation in some locations. This is highly dependent on local demand and ease of access.

Insect control: release of introduced insects to weaken or kill specific target plants has proven effective in certain noxious weed

Dimensions of Treatment Methods

situations. This is not applicable in plantation maintenance where precise timing and span of control—a variety of plants—is generally needed.

Pathogenic control and allelopathy: use of introduced pathogens and chemicals produced by plants to repel or inhibit competitors is still experimental in forest management.

Fire Management Program

Background

The fire management program within the Pacific Northwest Region developed early in the 20th century in response to a series of devastating wildfires. Initially, the stated policy was that all wildfires were to be controlled, regardless of cost, by 10 a.m. the day following discovery. The 10 a.m. policy remained in effect for 43 years. In 1978, the policy was rescinded as a result of increased awareness of fire economics and resource values. Today, the policy is to suppress all wildfires in a timely and safe manner. Suppression strategies are used which minimize both suppression cost and resource damage.

The fire program consists of activities undertaken for the protection of resources and other values from wildfire, and the use of prescribed fire to meet land and resource management goals and objectives. The Region spends approximately 26.4 million dollars annually to protect 26 million acres.

Protection activities include fire suppression, detection, and prevention, and fuels management. The Region has an extensive suppression organization comprised of smokejumpers, hand crews, helitack crews, air tankers, engine crews and fire overhead teams. Suppression forces can be mobilized and transported to remote locations throughout the Region quite rapidly.

Detection of wildfires is accomplished by a variety of methods. Fire lookout towers are still used on many Forests, although some are used only during periods of high fire danger. Ground patrols often are used during periods of high fire danger and aerial patrols are quite effective in inaccessible terrain. Recent detection advances include the addition of electronic lightning detectors. The electronic lightning detector, in conjunction with a computer, charts the location of known lightning strikes.

The purpose of wildfire prevention is saving lives and avoiding unacceptable losses to resources, property, and improvements. The Region coordinates with local, state, and other federal agencies to ensure that prevention efforts are successful. The prevention program is targeted for all forest users, both recreational and industrial.

Fuels management consists of planning and executing the treatment or control of living or dead vegetative material. Fuels are treated to provide cost-efficient resource protection and to meet land management objectives. Currently, about 200,000 acres are burned annually to manage fuels. Logging slash accounts for about 160,000 acres of the total acres burned.

The use of prescribed fire for the protection, maintenance, and enhancement of resource productivity is also a part of the fire management program. Fire can be used whenever practical and cost effective and when carefully planned and administered.

The following Acts contain legal requirements and authorities to plan and carry out activities to protect National Forest System lands and resources from fire:

1. Act of June 4, 1897 (16 U.S.C.551)
2. Bankhead Jones Farm Tenant Act, July 22, 1937
3. National Forest Management Act of 1976
4. Wilderness Act of Sept. 3, 1965
5. Endangered American Wilderness Act of 1978
6. The Clean Air Act Amendments of 1977
7. Clarke-McNary Act of 1924

The National Fire Management Analysis System (NFMAS) is used to determine fire suppression resource and program needs at the Regional and Forest level. The system provides a consistent budget analysis process for evaluating the efficiency and effectiveness of fire management programs. Direction for forest fire protection and use programs, appropriate to meet resource targets, is developed from resource management objectives and prescriptions through the forest planning process.

Site-specific fuels treatment alternatives are developed through the interdisciplinary process. A team of resource specialists evaluates a proposed activity and recommends needed fuels treatment. Treatment for protection purposes is based upon historical fire occurrence, expected fuel loading, expected fire risk, and expected fire hazard. Treatment methods are selected after careful evaluation of the site. Factors such as slope, stability, vegetation type, soil compactibility, erosion potential, and smoke management restrictions are considered prior to selecting a treatment method.

All methods of managing unwanted vegetation are appropriate for fire management activities. The Region's preferred method of managing unwanted residue is utilization. Opportunities to use residue are

Authority and Legal Requirements

Implementation

Methods

actively pursued before other methods are considered.

Other treatment methods are used if residue utilization fails to meet fire management objectives. Prescribed fire, the controlled use of fire under predetermined conditions, frequently is used to accomplish fire management objectives. Fire is used alone and in conjunction with other vegetation management methods such as yarding of unmerchantable material (YUM), machine and hand piling of slash, and machine crushing of slash. Use of fire requires close coordination between federal, state, and local agencies to ensure that impacts of smoke on air quality are minimized.

Chemicals used in fire management activities generally are limited to fire retardants and alumagel. Retardants are sometimes used for fireline on prescribed fires. Alumagel, a gasoline thickening agent, is used as helitorch fuel to ignite prescribed fires. Alumagel is also used to ignite large slash piles for disposal during wet weather conditions. Chemicals are seldom used to desiccate vegetation prior to burning.

Range Improvement Activities

The rehabilitation of deteriorated rangeland, protection of rangeland from degradation, or improvement of forage quality or quantity often requires manipulation of vegetation. Rangelands that become less productive due to overgrazing, or through natural changes in vegetation (succession) must be managed to meet the objectives established for them, i.e., increased livestock production, improved wildlife habitat, or watershed. These changes occur more rapidly in the absence of fire, and tend to lead to a dominance of shrubs over herbaceous plants, and an overall reduction in plant species diversity.

Range improvement activities are carried out primarily under provisions of the Federal Land Policy and Management Act of 1976 and the Public Rangelands Improvement Act of 1978. In recent years, vegetation management has taken place on approximately 2,000 to 4,000 acres of rangeland annually.

On National Forest System lands in the Pacific Northwest, grazing of domestic livestock takes place on approximately 6.7 million acres in 807 grazing allotments. Management of each grazing allotment is governed by an Allotment Management Plan, which identifies site-specific capabilities, grazing systems and intensities, and structural and nonstructural range improvement projects necessary to implement the selected grazing system. Planning for individual vegetation management projects (a form of nonstructural range improve-

ment) involves environmental and economic considerations, and is documented in an Environmental Assessment or Categorical Exclusion.

The most common targets of vegetation management activities are sagebrush, rabbitbrush and western juniper. These woody species may increase on rangelands where fire has been excluded or overgrazing has occurred. Growth of these species occurs at the expense of more palatable grasses and forbs. Control reduces the density of shrubs and trees, allowing increased production of more desirable herbaceous understory vegetation. Treatment may also be necessary to restore the productivity of disturbed meadows in forest zones, or abandoned fields on the Crooked River National Grassland.

Herbicides

Herbicides have been applied either from the air or ground to eliminate or control unwanted plant species such as sagebrush and rabbitbrush.

Mechanical

Plowing is used on productive sites to control small, shallow-rooted plants and prepare a seedbed. Chaining breaks plants off or pulls them over, and is accomplished by dragging a heavy anchor chain in a U-shape behind two crawler tractors. Crawler tractors with brush or dozer blades are used to push over and pile unwanted woody or brushy species.

Manual

Handcutting of shrubs and small trees is used on a limited basis.

Biological

Control of some species can be accomplished through the use of grazing animals such as goats.

Prescribed fire

Prescribed fires are the most commonly used form of vegetation control in recent years. Undesirable shrubs are effectively reduced while growth of palatable forage species is enhanced.

Methods

Noxious Weed Control Activities

Noxious weeds are defined by both federal and state laws. They are species of plants that cause disease or are injurious to crops, livestock or land, and thus are detrimental to agriculture, commerce or public health (PL 93-629, ORS 570.505, RCW 17.10). Many species are harmful to agricultural crop production, or are toxic to livestock that ingest them. Designation of species as noxious weeds can be made by either United States or state departments of agriculture.

The overall goals of noxious weed control activities are to limit the spread of such plants, reduce their numbers to a point where they cause no significant economic damage, and—where feasible—eradicate them. Control measures are conducted under authority of the Carson-Foley Act (PL 90-583) and the Federal Noxious Weed Act (PL 93-629). In recent years, acreages treated to control noxious weeds in the Pacific Northwest Region have ranged from approximately 3,000 to 10,000 acres per year.

On National Forest System lands, measures are taken to protect range values, wildlife habitat and sensitive plant species, as well as to reduce the potential for spread of noxious weeds to other ownerships. Since noxious weeds fail to respect state, administrative, or property boundaries, close coordination among federal, state and county agencies, as well as private land owners, is crucial to any effective program to control noxious weeds.

Control programs in the Pacific Northwest Region are conducted in cooperation with the Oregon Department of Agriculture, as well as individual counties or weed control districts in Washington State. The main distinction between the two states is that the Oregon Department of Agriculture has authority and responsibility for direct control measures, while in Washington control activities are carried out by the counties, since the state has no such legal mandate.

The level of threat posed by various species of noxious weeds depends on such factors as the degree of detrimental effects they can produce, their reproductive and dispersal capabilities, difficulty of control and overall distribution. Based on such considerations, particularly distribution and feasibility of control, the state of Oregon has developed a weed classification system. In Washington a noxious weed list is developed at least yearly through a hearing process by the state noxious weed control boards.

Infestations of noxious weeds are located either through surveys, or incidental location by Forest Service personnel trained in their identification, or Forest contractors or other Forest users. All infestations are considered undesirable, with the intensity and method of

control depending on a variety of factors. These factors include classification category, species biology, size of population, geographic location, potential for spread, other land characteristics or uses (such as presence of sensitive species, watershed values, and health risks/threats) and budget considerations. General priorities for control should be established at the Forest level through noxious weed management plans developed in coordination with the states and affected counties.

Herbicides

A variety of techniques have been used to apply chemicals to control noxious weeds, including aerial (helicopter and fixed-wing), ground vehicles, and backpack sprayers. One potential chemical technique not yet fully developed is the use of species-specific herbicides that inhibit photosynthesis.

Mechanical

Various mechanical techniques can be used to prevent seed production by noxious weeds; reduce their vigor; and, in some cases, eliminate them from a site. Mowing, cutting, or tilling may be appropriate, depending on target species, associated vegetation and terrain. In some woody species cutting once every two years may be adequate to prevent flowering and seed production, while some herbaceous weeds may need to be mown two or more times per season. For species which can spread vigorously from root stocks, mowing may lead to increases in population.

Manual

Hand-pulling, digging, and clipping have all been used to help control noxious weeds. Similar in many respects to mechanical techniques, these have generally been viewed as stop-gap measures to reduce seed production, and generally not used to eliminate populations, except where very few plants are involved. Hand pulling and digging will be of limited effectiveness if care is not taken to remove all root material. Otherwise, resprouting is likely to occur.

Biological

Biological control techniques involve the use of insects, mites, pathogens (such as fungi), grazing animals, and competitive plants.

Basically, host-specific insects and pathogens are used to control plant numbers and vigor, reduce seed production and limit spread. Especially when used in combination, such control agents may effectively eliminate noxious weeds from an area. However, their use

Methods

is limited to areas where noxious weed densities are sufficient to allow the build-up of populations of the biological control agents.

Collection and redistribution of insects increases effectiveness by supplementing natural rates of spread of the control agents. Several insects are currently in use for control of noxious weeds in the Northwest, and additional potential biological control agents are being tested. Grazing animals tolerant of the toxins produced by noxious weeds can be used in some cases to control weeds on a limited basis (for example, using sheep to consume tansy ragwort). In some areas, planting desirable competitive plants (grasses, legumes, etc.) may successfully reduce or eliminate noxious weeds.

Prescribed Fire

Fire can be used to kill annual weeds, to reduce woody shrubs and other perennials, and, in some cases, to prevent seed production. Burning has not been extensively used for control of noxious weeds, since its use is restricted to areas where weeds are dense enough to make the technique practical, and where environmental considerations such as air quality are not limiting.

Wildlife Habitat Improvement Activities

The principal objective of wildlife habitat improvement activities is to maintain an appropriate variety, amount, and distribution of habitat in order to maintain viable populations of native wildlife, fish and plants. Particular emphasis is placed on maintaining threatened, endangered and sensitive species.

Habitat improvement is accomplished through coordination and mitigation procedures related to various resource activities and through direct habitat improvement projects. Primary direction for these objectives is found in the National Forest Management Act of 1976, and USDA Departmental Regulation 9500-4.

To a large degree, objectives for habitat management reflect population goals established in coordination with state fish and wildlife agencies. The Sikes Act of 1960 establishes the basis for cooperative relationships with the states regarding wildlife management.

Vegetation management activities are conducted to benefit a wide variety of wildlife species—from rare plants and butterflies to grizzly bears—but the majority of such activities are designed to provide improved forage and browse for elk and deer.

Herbicides

While the use of chemicals to control vegetation for wildlife habitat improvement has been relatively limited, opportunities for such manipulation exist. Shrubs can grow so tall and thick that they fail to provide useable browse, and may also impede movement. Herbicides, applied either from the air or ground, can alleviate these problems in some cases. Similarly, herbicides have been used to restore forage productivity on elk range overrun by unpalatable weeds.

Mechanical

Various mechanical techniques are used to manipulate vegetation for wildlife, including chaining, cabling, crushing, and scalping. These techniques are most commonly used on brush, to induce resprouting for improved browse, to reduce competition to favor herbaceous forage or to create travelways for big game species that may have difficulty moving through dense stands of brush. Mowing has been used to control grasses to improve growth of violets that are essential to the life cycle of the Oregon Silver Spot butterfly, a threatened species.

Manual

Manual techniques (such as cutting back shrubs or trees encroaching on meadows), are used to control brush or other vegetation on a limited basis. Cutting is also prescribed to regenerate aspen stands, or pruning of trees or shrubs to improve production of fruits and berries.

Biological

Controlled use of domestic grazing and browsing animals has been employed to control shrubs, and to reduce unpalatable portions of herbaceous forage so that later regrowth can be consumed by wildlife.

Prescribed Fire

Fire is by far the most commonly used tool. Underburning in forested stands to improve forage is the most widespread use, but fire is also employed to control or rejuvenate brush, to maintain or create meadows and other openings, to remove slash impeding movement of big game animals, and as part of site preparation prior to planting forage.

Methods

Table G-1

**Annual Wildlife and Fisheries Report:
Estimated Consumptive and Nonconsumptive
Wildlife and Fish User Days (WFUD's)
(Fiscal Year 1984)**

Forest Name	Hunting	Fishing	Nonconsumptive	Total WFUD's
Colville	152,806	138,826	7,037	298,669
Deschutes	134,133	503,438	18,625	656,196
Fremont	51,786	92,010	2,483	146,279
Gifford Pinchot	286,752	464,926	64,967	816,645
Malheur	109,619	24,506	4,140	138,265
Mt. Baker-Snoqualmie	284,269	351,825	82,350	718,444
Mt. Hood	146,162	472,604	100,967	719,733
Ochoco	124,832	103,254	10,759	238,845
Okanogan	79,376	118,045	24,828	222,249
Olympic	69,183	74,523	1,656	145,362
Rogue River	34,191	80,956	434	115,581
Siskiyou	64,452	144,005	10,658	219,116
Siuslaw	86,490	259,140	19,447	365,077
Umatilla	1,100,093	36,050	26,254	1,489,398
Umpqua	87,839	287,718	10,347	385,904
Wallowa-Whitman	390,379	235,189	84,007	709,575
Wenatchee	418,063	538,600	72,002	1,028,665
Willamette	172,897	707,720	61,244	941,861
Winema	54,335	53,798	828	108,961
Subtotal WFUD's	3,847,657		4,450,690	
Total WFUD's, hunting and nonconsumptive uses: 8,298,347				

(Note: a WFUD for hunting could be the equivalent of one person hunting for 12 hours, or 12 persons hunting for 1 hour. Use expressed in wildlife and fish user days (WFUD's) is determined from the best source of information available for wildlife- and fish-oriented recreation; whether from the recreation information management data base (RIM), from the states, or from other sources.

Table G-2

**From 1984 Annual Wildlife and Fisheries Report
Regional Totals**

<i>Species</i>	1983		1984	
	<i>Population</i>	<i>Harvest</i>	<i>Population</i>	<i>Harvest</i>
bighorn sheep (desert)	—	—	—	—
bighorn sheep (other)	582	9	585	10
bison	—	—	—	—
black bear	18,332	1,572	17,770	1,310
black-tailed deer	222,133	18,038	210,675	17,641
caribou	6	0	6	0
Dall sheep	—	—	—	—
elk, Rocky Mountain	69,595	15,019	63,622	12,941
elk, Roosevelt	29,494	3,166	28,993	2,831
grizzly bear	4	0	4	0
moose	45	0	49	0
mountain goat	4,471	198	4,097	224
mountain lion	1,867	120	1,928	107
mule deer	217,570	25,123	190,465	22,574
pronghorn antelope	2,080	125	2,160	126
turkey	1,849	38	2,353	25
white-tailed deer	15,665	2,095	16,040	2,093
wild boar	—	—	—	—
wolf	2	0	2	0

Recreation Maintenance

The basic mission of this program is to provide outdoor recreation opportunities for the Nation. The objectives for publicly managed recreation opportunities, which include developed recreation sites, are:

- 1) To maximize opportunities for visitors to know and experience nature while engaging in outdoor recreation;
- 2) To develop and manage sites consistent with the available natural resources to provide a safe, healthful, esthetic, nonurban atmosphere; and
- 3) To provide a maximum contrast with urbanization at National Forest sites.

Forest recreation opportunities are classed as developed recreation, dispersed recreation areas, or special interest areas. Developed recreation refers to specific sites where facilities have been provided such as campgrounds, picnic areas, boat docks, or ski lifts. These sites are generally easily accessible and designed to provide a modern recreation experience in a natural setting. Dispersed recreation describes the use of lands and water bodies throughout the National Forests, where recreation opportunities such as backpacking, hunting, nature studies, primitive camping, or cross-country skiing are available. Minimal improvements such as parking and sanitation facilities at trailheads are provided for dispersed uses. Special interest areas are generally unimproved and have been designated for a specific value such as a geologic, scenic, historic, or botanic interest area.

Within the Pacific Northwest Region there are over 1,900 developed recreation sites, including permittee operations and privately owned sites within National Forest boundaries.

Vegetation management is part of the operation and maintenance of developed recreation sites. Vegetation management is used to provide for public safety, reduce fire hazards, improve visibility and access, and to control poisonous plants.

Forest Service Manual direction requires that a vegetation management prescription be prepared for each recreation site. The primary objective of the prescription is to create and maintain a natural-looking environment.

In general, these prescriptions call for trimming and removal of grass, brush, or trees necessary to allow the site to be used safely, while retaining over-story and understory cover. This includes trimming or removing vegetation along site access roads for adequate site distance, around signs and other facilities, and in individual camp or

picnic sites. Where necessary, direction is given for control of poison oak or other toxic plants. Hazard trees are removed when necessary. Vegetation surrounding (or interspersed among facilities within) sites is retained for a natural appearance. Brush removal is also prescribed to maintain cleared ski runs.

Developed sites are inspected annually to note any deficiencies, including safety hazards and vegetation treatment needs. Such inspections are documented in Ranger District records, which contain base data used to develop annual site operation and maintenance plans.

Techniques presently used in developed recreation sites are primarily manual and mechanical. In campgrounds and picnic areas, hand-held implements such as brush hooks, scythes, saws, and "weed eaters" are used to trim brush, and mowing machines may be used on roadside strips. Brush is either scattered or hand-piled, or piled by small tractors, and removed, burned, or chipped. When tree removal is necessary, the technique causing the least impact to the environment is used for yarding and brush disposal.

Tractors, along with manual methods, are used for brush removal and disposal to maintain cleared ski runs.

Facilities Maintenance

A facility is a single or contiguous group of improvements that exists to shelter or to support Forest Service programs. A facility may be a ranger station compound, lookout tower, leased office, work center, separate housing area, visitor center, or research laboratory. There are approximately 200 of these sites in the Pacific Northwest Region.

Facilities are managed to provide cost-effective, safe, functionally-efficient buildings and related improvements for conducting the work of the Forest Service. Vegetation management is performed to provide safe working conditions; provide for protection of materials and property; and to provide an aesthetically pleasing appearance for the facility.

Each administrative site has a site development plan that includes a landscape management plan or planting plan. The landscape management plan delineates the areas where vegetation will be maintained or controlled, and prescribes appropriate treatments, including manual, mechanical, and chemical methods. Chemical methods at this time are limited to fertilizers and pesticides that are used on lawns and shrubs to enhance growth.

Rights-of-Way Maintenance

Rights-of-way maintenance includes controlling vegetation along (and within) highways and roads, land lines, trails, utility corridors, and railroads. Historically, vegetation control programs for rights-of-way maintenance have included the full range of options—manual, mechanical, biological, thermal, and chemical.

USDA-Forest Service Roads

Forest roads are of primary importance for the protection, administration, and utilization of the National Forests and other areas administered by the Forest Service, or for use and development of resources upon which adjacent communities are dependent.

The National Forest Management Act of 1976 requires that “roads constructed on National Forest System lands shall be designed to standards appropriate for the intended uses, considering safety, cost of transportation, and impacts on land and resources.” Roadside vegetation management is used to protect this investment and provide safety for the users in concert with the roads intended use. (National Forest Management Act, Section 8 (c).)

Current maintenance standards for Forest roads are defined in Forest Service Handbook 7709.15. Maintenance Levels 1 through 5 determine the intensity of maintenance, including roadside vegetation management. Maintenance Level 1 does not require vegetation management except as necessary to protect the investment. Maintenance Level 2 requires brushing to provide passage for high clearance traffic. Maintenance Level 3 requires brush control to provide for safe sight distance. Maintenance Levels 4 and 5 requires brush control to be accomplished on a scheduled basis for safe sight distance and for appearance. Approximately 29 percent of the total road system is in Maintenance Levels 3 through 5, and the remainder is in Maintenance Levels 1 and 2.

Forests currently use these standards in conjunction with established road logs, condition surveys, Road Management Objectives, and periodic maintenance plans to determine when, where, and how vegetation management will take place on Forest Service roads. Economics, environmental considerations, the current restriction on herbicide use in the Pacific Northwest Region, and public concerns all have an influence on the method of vegetation management selected. Line Officers at the Forest level (District Rangers or Forest Supervisors) generally make the final decision as to what method will be used.

The following methods have been used in the past with varying degrees of success:

1. Manual techniques have received limited use, but are effective in certain instances. These are used mainly on the inside of curves where mechanical mowers will not reach; in areas where brush is sporadic and it is not cost effective to run a mechanical mower down a road; and for doing hand pulling or grubbing of certain target species.
2. Biological methods are mostly confined to planting and fertilization of grasses. Biological methods have been considered experimental in the past, but are gaining favor as a preventive measure rather than a curative treatment.
3. Mechanical techniques, used extensively by the Forest Service, are effective treatment methods. Mechanical brushing is performed with tractor-mounted flail type mowers, rotary type mowers, or sickle bars. However, with fast growing target species, mowing must be done periodically, and this usually increases the density of resprouting target species, adding to the overall problem. Forest Service personnel who perform this work are licensed equipment operators.
4. Thermal techniques are seldom used. Piling and burning of right-of-way brush is occasionally used but has a high cost. Backpack flame throwers have been used on a very limited basis to control vegetation adjacent to asphalt pavements.
5. Chemical methods are effective when used properly. However, rising social and environmental costs—along with the current injunction on using herbicides—have given rise to using less chemical treatments and exploration of other nonchemical alternatives. Before the injunction, herbicides were generally applied along roadsides using backpacks, mobile booms, or hand wands. All Forest Service applicators were licensed by the States (Oregon or Washington) where the roads are located.

From a practical standpoint, methods 1,2,3, and 5 above are all viable options. There are approximately 87,900 miles of Forest Service roads in the Pacific Northwest Region. These range from very dry conditions on the East-side of the Cascades (where little roadside brushing is required) to very wet conditions on the Coast (where heavy, dense, fast-growing brush is common).

In addition to controlling vegetation along rights-of-way for maintenance, it is also necessary to handle unwanted vegetation along newly constructed roads. The Pacific Northwest Region annually constructs approximately 600 miles of road each year. Options for disposal of clearing debris include windrowing, scattering, burying,

chipping, piling and burning, disposal in cutting units, removal, piling, and placing on embankment slopes.

Highways Highways affected by the Forest Service vegetation management program include the public vehicle transportation system of the Department of Transportation for Oregon and Washington; other Federal agencies such as Bureau of Land Management; and specific counties in Oregon, Washington, and California.

Vegetation management along those routes is designed to meet responsible agency maintenance objectives. These include:

- perpetuating the transportation facility to serve its intended purpose;
- protecting the investment;
- protecting the environment;
- providing for user safety, economy, access, and convenience; and
- meeting all applicable air and water quality standards.

Public vehicle transportation system agencies maintain approximately 4,300 miles of highways within the National Forests of Oregon and Washington. Historically, roadside vegetation along these roads has been controlled using manual, mechanical, biological, and chemical methods.

Utility Corridors A number of public and private utilities have rights-of-way through the Region's National Forests. Power line utilities have above-ground and buried cable line rights-of-way and access roads, along with substation property and power plant sites. These utilities maintain approximately 25,800 acres on National Forest land.

Communication utilities have transmission rights-of-way, access roads, wave guide routes, communication towers, and exchange (relay) stations. The 50-plus communication network utilities annually maintain approximately 1,500 acres on National Forest land.

There are also a number of agencies and private utilities with permits allowing the transport of water in open channels or pipes for use in farm irrigation or at electrical power plants. There are 493 waterway permits involving approximately 1,000 acres of rights-of-way. About 25 percent of the total is ditches.

Vegetation control programs are used to keep trees and other tall vegetation from growing into conductors, thus preventing power outages and possible forest fires. Vegetation is also controlled along access roads and along waterways (irrigation ditches) to control aquatic and noxious weeds.

Manual, mechanical, and chemical methods are all used to control vegetation. In metropolitan and urban areas mechanical and manual methods are the primary treatment for vegetative control due to ease of access. In forest areas the lack of access for mechanical equipment often results in a strong preference for chemical treatment.

Over 90 percent of the waterway treatment is by manual or mechanical methods.

Railroad transportation systems have rights-of-way, access roads, crossings, and communication lines (including towers) throughout National Forest lands in Oregon and Washington that require annual vegetation control to prevent train-caused fires; to maintain the roadbed and eliminate safety and nuisance problems for railroad personnel; to maintain ditches and other drainage structures; and to provide visibility at railroad crossings.

Annually, railroads (there are 17 permits) use manual, mechanical, and chemical methods to control vegetation on roadbeds and fire breaks along 105 miles of right-of-way.

Railroads

Throughout the Pacific Northwest Region of the Forest Service there are approximately 15,000 miles of established trails. Vegetation along these trails is controlled to protect the investment, to provide a reduction of poisonous plants, and to eliminate branches and stems for user safety.

Annually, the Region performs vegetation management on approximately 3,200 miles of trail. Vegetation is cleared within four to eight feet of the trail tread, depending on the trail standard.

Nearly all vegetation management is accomplished using manual methods. Approximately one percent of the total vegetation control effort has utilized chemical treatments in the past.

Trails

“Land line location” is a Forest Service land surveying program that identifies, marks, posts, and maintains legal property lines and administrative boundaries where property rights and/or management activities require accurate field delineation because of a legal requirement (Forest Service Manual 7151.06 6.).

Except for lines posted for subdued visibility and areas where vegetation regrowth will quickly obscure the line, the property line is to be cleared of small trees, brush, and debris for a distance of about two feet on each side of the line. When an offset method of survey is used, only the property corners are posted and cleared. Hand methods (for example power saws or machetes) are almost always used.

Maintenance is performed on a periodic basis. The maintenance interval is dependent on local conditions, but should never

Land Line Location

exceed ten years.

There are approximately 12,000 miles of lines that have been located and established by an acceptable official survey of record that should be periodically maintained. For fiscal year 1987, it is estimated that 150 miles of line will be maintained.

Tree Genetics Program

Background

The demand for a diversity of forest products is increasing, yet the land base for producing timber products is decreasing. Thus, there is a need to improve productivity on the remaining acres. The genetics program was developed as an integrated Regional strategy to fill this need in both the long and short run.

The program reaches out through cooperators to both other public and private lands as a further measure to assure a continued flow of timber products.

The Region spends approximately 7 million dollars annually on genetics and related activities. There are 252 seed orchards and 262 evaluation plantations totaling 5,298 acres. A number of superior parent trees have been selected and are being continually evaluated. All the Region's National Forests have geneticists or persons with appropriate expertise who administer the program locally. A local tree improvement plan provides guidance and coordination with Regional goals.

Purpose and Objectives

The purpose of the genetics program is to increase yields of high quality forest products. Individual trees that exhibit specific outward characteristics are selected as parent trees to provide seed for future generations or crops.

Three characteristics or traits are of concern: rapid growth with high quality wood; resistance to insects and disease; site specific adaptation; or some combination of the three. For example, an individual sugar pine tree may be selected as a parent because it is resistant to blister rust and has a rapid diameter growth rate. The intent is to capture the genetic material responsible for controlling the desired traits.

Implementation

District and Forest personnel are responsible for the implementation of the program. Area geneticists coordinate among several forests. Most activities are centered around parent tree selection, installing and maintaining evaluation plantations and operating seed orchards. Vegetation management activities are applied to enhance seed production and tree vigor; facilitate operations; and reduce fire hazard and

animal habitat. Survival and unimpaired early growth are also essential. A uniform environment around each parent is necessary to minimize the effects of the environment and maximize the expression of its heredity. Evaluation of the parent is most reliable under these conditions.

The intensity of the vegetation control is dependent on site factors such as slope steepness, aspect etc.; and on soil variation, including erodibility, infiltration rate, and chemical composition. Site degradation is not tolerated.

Site-Specific Considerations

Research Program

The purpose and objective of the research program is to develop knowledge and technology to enhance the economic and environmental values of forest and rangeland. The program seeks better ways to use forest and rangeland resources by developing technology to reduce costs, increase productivity, and protect environmental quality.

Projects involve biologists, economists, engineers, social scientists, and those in other disciplines, often in cooperation with universities, State agricultural experiment stations, other U.S. agencies, and foreign countries.

The knowledge is used by the National Forest System at all levels to assist in setting policy and to provide information for site specific projects and long term planning.

Forest Service research is authorized under the Forest and Rangeland Renewable Resources Research Act of 1978 (92 Stat. 353). This act complements the policies and direction set forth in the Forest and Rangeland Renewable Resources Act of 1974 (16 U.S.C. 1641) directing the Forest Service to obtain, analyze, develop, demonstrate, and disseminate scientific information about protecting, managing, and utilizing forest and rangeland renewable resources.

The Pacific Northwest Station operates ten field laboratories located in Alaska, Oregon, and Washington. It employs approximately 350 people (about a third are scientists) with an annual budget of approximately 16 million dollars. An additional four million dollars helps to fund cooperative projects with agencies, universities and private corporations from around the country.

Background

Research projects are commonly based on National Forest System needs. There are a significant number of projects that deal with vegetation management techniques, concepts and environmental effects.

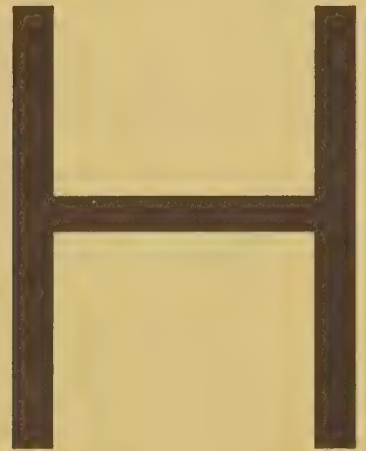
Implementation

Information needs on vegetation management techniques and effects have been increasing, thus there is a concurrent need for a full array of vegetation management research.

In cases where vegetation management is the subject of study, levels of control are determined by the study design and the specific objectives of the study. However, vegetation management is often needed to maintain research plots. Corners, markers, and access need to be clearly visible and functional and control measures are applied accordingly.

Appendix H

Human Health Risk Assessment (Qualitative)



Appendix H

Human Health Risk Assessment (Qualitative)

Section 1. Qualitative Risk Assessment

Section 2. Data for Analysis of General Systemic Toxicity

Section 3. Data for Analysis of Mutagenic and Carcinogenic Toxicity

Section 4. Data for Analysis of Reproductive and Developmental Toxicity

Section 5. Data for Analysis of Immunotoxicity and Neurotoxicity

Section 6. Data for Evaluation of Human Epidemiology

Appendix H
Human Health Risk
Assessment
(Qualitative)

Section 1
Qualitative Risk
Assessment

Qualitative Risk Assessment

Why a Qualitative Risk Assessment?

Human health risk management by the U.S. Forest Service has been troubled by strongly conflicting views on the potential for health risk in vegetation management. Various sides embrace very different scientific judgments and policy choices on both the risk assessment and risk management levels. The debate, however, has not generally recognized these differences, and has been argued on an item by item, right vs. wrong basis.

Part of the problem is that there is a great deal of uncertainty in the data on health effects of herbicides. This uncertainty has to be bridged with human judgment. These controversial decisions have not been clearly presented, and perceived health risks have become a major focus of public input and concern.

However, by more clearly stating the various unknowns, and the limits of our judgments, we hope to clarify these issues. Thus, the concept of a qualitative risk assessment. We hope to put equal emphasis upon the information the data contains and the quality of that information. We will avoid stating numerical risks where we do not have confidence in the numbers.

Risk assessments face two types of information gaps—gaps in test data and gaps in testing theory. Actually, it is the interaction of the two that makes our work so uncertain. The lack of accurate measurement tools makes the task of gathering information much more difficult.

It is important to understand that the biological models we use to measure health risks are very simple compared to the complexity of life. When we try to measure the effect of a chemical on any living organisms, individual variability is extremely large.

Because of this biological variation, a dose that may seriously affect one test animal may produce no observable effect on another of the same species. To deal with this individual variability, we have developed methods of statistically measuring the probability of observing an effect in a test population exposed to a chemical.

In an effort to evaluate the cancer potency of various chemicals, a group of researchers (Gold et al. 1984) reviewed over 3,000 long-term animal experiments for 770 different chemicals. They evaluated the dose that would cause 50 percent of the test animals to contract cancer (Tumor Dose 50), and found a ten million-fold variation in potencies between the various chemicals, and a thousand-fold variation within some individual chemicals.

The Debate

Looking at the Uncertainty

This information tells us that toxicity studies are useful because the variation in potency in tests for a particular substance is small compared to the variation between substances. In other words, we can make some distinction between the carcinogenic potency of various chemicals.

However, this also demonstrates that the results of different studies evaluating the cancer potency of a single chemical could disagree by a factor of 1,000. This wide range might be due to species differences, different methods of administering the chemicals, or other factors. Whatever the reason, we must use caution in trying to quantify specific toxicity using published studies.

Two Types of Errors

Much of the disagreement around human health risk assessment is based upon how to weigh studies showing differing results. Because there is biological variability, it is unreasonable to expect that all test results will agree. Statistical methods allow for a certain number of false conclusions.

When testing a substance for toxicity, it is usual to start with the hypothesis that assumes no effect—the ‘null’ hypothesis. Statistical methods are applied to the test results to determine if the null hypothesis is consistent with the data. If the null hypothesis is not too discordant with the data, it cannot be rejected, and the study is considered negative. If it is discordant with the data, then it is rejected for an alternative hypothesis, and the test is considered positive.

Thus, when we use statistical tests to determine whether or not an effect occurs (such as whether or not a substance is a carcinogen), there are two types of errors that can be made. First, the null hypothesis may be rejected, and the conclusion made that the chemical does cause cancer when, in fact, it does not. This is called a false positive.

The second type of error occurs when the null hypothesis is not rejected. The test is then considered negative, and the conclusion may be made that the chemical does not cause cancer, when it actually does. This is called a false negative.

Both false positives and false negatives are extremely important types of errors, but their treatment by the scientific community has been inconsistent. Most authors report the probability of making a false positive conclusion, but not the probability of a false negative conclusion. This may lead readers to discount the importance of false negatives.

On the other hand, those involved in developing risk assessment and risk management policies frequently consider false negative conclusions more costly than false positives. For example, Lave and Omenn (1986) make the assumption that, in evaluating chemicals for

carcinogenicity, a false negative may have ten times the social cost of a false positive.

A study is called significant when the probability of the the test data occurring under the null hypothesis falls below some preset level. By convention, this level is five per cent. When a study is considered positive, the statistical probability of this conclusion being false (a false positive) is the level of significance of the study. The lower the probability of a false positive, the stronger the significance of the study. This probability, often expressed as the 'p' value, is a universally reported statistic. Only when a study reaches the preset 'p' value criteria is it considered to support the positive conclusion (a single study does not prove a conclusion—it can only support it).

When the 'p' value of a study is greater than 0.05 (or 5 percent), by convention it is considered "not significant" and does not support the rejection of the null hypothesis and acceptance of an alternative hypothesis. This is not, in itself, evidence that there is no effect. In fact, this is where the importance of the second type of error—the false negative—comes in.

Significance

It is possible, and at times probable, that a true effect will not meet the criteria for significance. It is important to know the probability of being able to reject the null hypothesis when there is a true effect (probability of "seeing" the effect). This probability is dependent upon the design of the study as well as the strength of the actual effect being measured, and is called the power of the study.

Power

The power of a study is always less than one (there is no certainty of seeing the effect). The probability of missing the effect (coming to a false negative conclusion) is one minus the power. While there is no strong convention on what an acceptable level of power is, 80 percent is generally considered strong. This would mean that there is a 20 percent chance of missing a true effect. The power of a study is usually considered part of the study design rather than part of its conclusion, and is often not reported in the scientific literature.

In order to appreciate the function of the power of a study, it is necessary to understand that studies do not measure safety (the total lack of effect), but only levels of effect. The smaller the effect, the harder it is to measure. A study designed with a good level of power to detect a doubling of risk, may have virtually no chance of seeing a true increased risk of only ten percent.

Thus, the power of a study is dependent upon the level of effect you are interested in measuring. The most important parameter of a study design in determining its power is the size of the test popula-

tion. If you are interested in observing an effect that occurs in a very low percentage of the population, then you must test a very large number of subjects. But it is very expensive to design large, high powered studies for low level effects. Thus, the probability of false negatives is generally much higher than the accepted preset five per cent level for false positives.

Interpreting False Positives

What do false positives mean in terms of public health and the decisions facing the Forest Service? A false positive means that a chemical will be considered dangerous when, in fact, it is not. It will mean that the chemical may not be used, or may be used in reduced amounts and in restricted circumstances.

It may also mean that alternative ways of doing the job may be chosen. These alternative methods may have their own risks. Thus, using a less-tested chemical or alternative method for one that is well-tested and demonstrated to have some toxic effects, may not reduce true health risks.

What is of particular concern to those who believe chemical use is beneficial is that a single chemical may be tested many times, and any ONE of these tests, if significant, could be used to label the chemical as toxic. Clearly, as the number of tests increase, the probability that any one of them may be significant simply by chance (false positive) goes up. For this reason, supporters of chemical use suggest that consistent positive results be required to determine toxicity.

Interpreting False Negatives

What do false negatives mean in terms of public health and the decisions facing the Forest Service? A false negative means that a chemical will be considered safe when, in fact, it is dangerous. It will mean that the chemical may continue to be used in situations where people are exposed, and where it may be harmful to their health.

It also means that alternative ways of doing the job may not be fully explored. These alternative methods may have reduced risks. Thus, opportunities for doing the job in a safer way may be overlooked.

What is of particular concern to those who are worried about the extent of chemical use is that a single chemical may only be tested in a few minimally adequate tests—tests with high likelihood of producing false negatives. If these tests do not show a significant effect, then the chemical is labeled as not toxic.

Finally, those concerned about use of chemicals point out that unknown situations are frequently considered safe situations. Use of chemicals is not generally restricted without clear evidence of harm,

and many chemicals are used with little or no information about their toxicity.

The degree of uncertainty for yes-no answers is quite high. Consider the paper sponsored by the International Commission for Protection Against Environmental Mutagens and Carcinogens (Clayson & Krewski 1986):

“For example, with an agent affecting as much as 1% of the population, there is a better than even chance of observing no responses in a sample size of $n=50$.”

Also:

- “with 50 animals per group, the false negative rate can be more than 50% with an agent causing an excess risk of 10% over and above background rate.”

The authors continued by pointing out that to detect low level effects with any reliability could require thousands of test animals per experiment.

One method of avoiding this uncertainty is the use of upper 95 percent confidence intervals. This is a statistical technique for answering the question: What is the largest effect—with 95 percent certainty—that would occur after a given exposure.

Clayson and Krewski point out, however, that when this method is applied, it can have very conservative results. They give a hypothetical example of testing 50 animals with distilled water and observing no adverse health effect. Nevertheless, using the upper 95 percent confidence limit approach, the authors calculated that a maximum:

“dose of 833 ppb (parts per billion of distilled water in the food) would be required in order to limit the population risk to one in a million.”

The statistical methods discussed above are primarily used in the analysis of data from animal studies. Additional uncertainty arises from the extrapolation of these results to humans and to the lower doses humans are likely to encounter.

The way animals and humans metabolize various chemicals can be quite different, and may mean that the test results in one have no direct application to the other. In addition, test animals are generally selected for being healthy, and are maintained in otherwise healthy environments. These conditions do not apply to the general human population.

Questions on how to relate animal doses to humans, and which

Looking at the Degree of Uncertainty

Additional Gaps in Testing Theory

animals are the best predictors of human toxicity, have not been completely resolved.

Major concerns exist around the interactions between various chemicals. A mixture of chemicals is generally considered to have a toxicity equal to the sums of the toxicity of the various chemicals in the mixture. This is not necessarily true. Many chemicals interact with each other. Mixtures of chemicals may be either less toxic, or more toxic than the sum of their parts. This may be due to chemical to chemical interactions, or the altering of the host susceptibility to one chemical by another. Current testing methods seldom take this into account.

There are also considerations about various types of toxicity and our ability to test them. For example, standard test protocols have not been developed for neurological and immunological toxicity. Concern has also been expressed about the problem of hypersensitive individuals—people who are much more reactive to chemicals than would be predicted by general population reactions. Hyper-sensitive individuals are not accounted for in statistical tests which are based upon normal population distributions. One type of hypersensitivity is the allergic reaction, but other types of hyper-sensitivity also exist, and are poorly measured and poorly understood.

The above examples clearly indicate limitations in current methods of testing for toxicity. A manager for regulatory response for a chemical producer, in a review article on calculating carcinogenic potency (Barr 1985), stated:

“A completely acceptable method of estimating relative or absolute potency values relevant to humans has not yet become available. The nearest approximation is the upper limit on risk which can be estimated from epidemiological data.”

Gaps in Test Data

Data gaps exist which are not based upon gaps in theory, but on gaps in experiments. No matter how good or bad experimental methods may be, they cannot provide answers unless the chemicals of concern have been tested by currently acceptable methods.

Unfortunately, many of the chemicals being reviewed by the Forest Service have not been sufficiently tested. Furthermore, many methods available at the time of the initial testing do not meet current standards for acceptable test procedures.

This can be most clearly seen in the current review of pesticides being conducted by the state of California under the Birth Defect Reduction Act (SB 950). Reviews were available from California on 13 of the 16 herbicides being considered. Many of the tests reviewed were not considered to be of adequate quality.

Finally, when testing pesticides, most chronic tests do not use the full formula, but test only the active ingredient. However, a high proportion of most formulas are made up of the so called "inert" ingredients. These "inerts" are often neither chemically nor biologically inert and may have substantial toxicity themselves.

Inert ingredients are generally not reported on labels or safety sheets. The Environmental Protection Agency reports that many inerts have not been tested for toxicity, and there is virtually no information on possible interactions within these mixtures.

Some limitations of toxicity testing and rating have been presented. These limitations, particularly as applied to the 16 herbicides being evaluated by the Forest Service, are considerable. This uncertainty becomes apparent by doing a qualitative risk assessment.

In Summary

Appendix H
Human Health Risk
Assessment
(Qualitative)

Section 2
Data for Analysis
of General Systemic
Toxicity

Evaluation of Herbicide Chronic Toxicity Information Base Used for Draft EIS

An evaluation was made of the information/data base cited in support of the chronic toxicity thresholds ("lowest systemic NOELs") contained in the "Supplement to the Western Oregon Program—Management of Competing Vegetation Draft Environmental Impact Statement," (USDI-BLM, February, 1986).

For this evaluation, only studies involving repeated exposures of several weeks to lifetime durations were considered. Information and data on the general toxic effects of the sixteen herbicides cited in the draft EIS referenced above was compared with that contained in several other information sources.

Because chronic toxicity test studies of pesticides are most often not reported in the open literature, very few useful original reports were available for examination. Hence, most of the information used for comparison was from secondary sources which had had access to original contract reports submitted to regulatory or other agencies. Therefore, the approach was to examine the data cited in each review or report examined, and to compare the findings with respect to consistency among the reports and specifically with respect to the conclusion reached in the Draft EIS.

The overall quality of the information was judged on the basis of apparent thoroughness of the studies (a range of doses including no effect; sufficient duration (minimally three months or greater); whether multiple species had been tested; identification of the most sensitive effect or target site); and general quantitative agreement on the NOEL among the studies, and with the draft EIS specifically.

A qualitative evaluation of the toxicity information available was expressed according to the following rating scheme:

I = Inadequate to make judgement concerning a chronic systemic NOEL (e.g., wide variation in values found in different studies, target effects/sites not identified, limited time, doses or species);

M = Minimally adequate to make a judgement (e.g., two or more reports with a range of doses, which agree in general on NOEL, but which give little information on nature of effects and with limited confidence that the toxicity has been well characterized).

A = Adequate (numerous studies have been conducted, driving target effects clearly identified in more than one study, and quantitative values for NOEL agree well).

This evaluation did not consider effects of teratogenicity, mutagenicity, carcinogenicity, neurotoxicity, or immunotoxicity, as these are evaluated separately. Of the 16 herbicides for which chronic toxicity information was evaluated, three (2,4-D, 2,4-DP, and picloram) were judged to have adequate information that could be used for risk assessment with confidence. Six were judged to have minimally adequate information (amitrole, asualam, atrazine, bromacil, dalapon, hexazinone, and simazine), but more complete information or additional studies would enhance confidence for risk assessment. For four herbicides (fosamine, glyphosate, tebuthiuron, and triclopyr), the information available for this evaluation was considered borderline between minimally adequate and inadequate. Essentially, this translates to a low level of confidence in the NOEL conclusion. For two compounds (dicamba and diuron), the information cited in the Draft EIS or available for review from these sources was inadequate to judge suitability for risk assessment.

It is important to note that these evaluations of adequacy of information refer to the information contained in the Draft EIS or available from other, generally secondary, sources of information supplied. There may well be additional information extant which has not been made available for examination. A comprehensive search of the open literature for chronic toxicity studies is beyond the scope of this project. However, quite frequently routine chronic toxicity studies are not reported in the peer-reviewed literature.

Given the limitations of this effort, it is of interest that the NOEL estimated after examination of several information sources do not differ greatly from those reported in the Draft EIS, although the basis for the EIS values is clearly underdeveloped in the Draft.

DRAFT - 6/5/87

GENERAL TOXICITY AND SELECTED
ORGAN EFFECTS OF CHRONIC AND
SUBCHRONIC EXPOSURES TO HERBICIDES

AMITROLE

<u>Source</u>	<u>Effects</u>	<u>Rt</u>	<u>Sp</u>	<u>Dose-schedule-duration</u>	<u>NOEL</u>	<u>LOEL</u>	<u>Notes</u>
EPA 1985a	thyroid effects	F	R		0.5 ppm 0.025 mg/kg/day		
Cal one liners (no author, 10/83)	thyroid effects	F	R	13 weeks	0.5 ppm	2 ppm	
Hazleton Labs 1959	thyroid effects (50 ppm) fatty liver changes (100 ppm)	F	R	0, 10, 50, 100, 500 ppm for 26 weeks	10 ppm		"unacceptable"
Hazleton Labs 1960	dec. wt. gain; enlargement/ congestion of thyroid		F	28 days		<250 ppm (LDT)	
Bayer 1978	dec. food cons.; wt. gain inc. mortality	F	H	0, 1, 10, 100 ppm	10 ppm		oncology study "unacceptable"
Bayer 1978	dec. maternal wt. gain	G	W	0, 4, 40, 400 mg/kg/day days 6-18 of gestation	4 mg/kg/day		"acceptable"

ASULAM

Source	Effects	Rt	Sp	Dose-schedule-duration	NOEL	LOEL	Notes
EPA 1984b	fatty deposits in liver	F	R	90 days	2000 ppm 100 mg/kg/day		
EPA 1984b		F	D	6 mos.	60 mg/kg/day		
EPA 1985d		F	R	107 wks.	50 mg/kg/day		
May and Baker LTD. 1968	myocarditis; kidney lesions; testicular abnormalities	F	D	0, 50, 500 mg/kg/day 90 days	>500 mg/kg/day		
May and Baker LTD. 1970	fatty liver	F	R	0, 16, 80, 400, 2000, 10,000 ppm 90 days	2000 ppm	10,000 ppm	60% Asulox
May and Baker LTD. (no date)	inc. thyroid, body wts.	F	D	0, 60, 300, 1500 mg/kg	60 mg/kg	300 mg/kg	"guideline"
IBT 8/75 651-05129	inc. relative liver wts.	F	R	2 yrs.	400 ppm (LDT)	2200 ppm	1 year interim report
Pathology Lab Rhodia, Inc. #CH-2, 7/78	inc. thyroid, heart, kidney wts.	F	M	0, 1500, 5000 ppm 18 mos.	<1500 ppm		oncology study "guideline"

ATRAZINE

Source	Effects	Rt	Sp	Dose-schedule-duration	NOEL	LOEL	Notes
EPA 1984c		F	D	2 years	150 ppm 3.7 mg/kg/day		
EPA 1984c		F	R	3 generations	100 ppm 5mg/kg/day (HDT)		
Suschetet et al., 1974	dec. growth, food cons.; dec. relative, absolute kidney/liver wts.; altered N, Ca, excretion.	F	R	0, 100, 500 ppm or 0, 5, 25 mg/kg/day	none established		
EPA 1984		F	D	0, 15, 150, 1500 ppm 2 years	150 ppm or 3.75 mg/kg/day		used to calculate margins of safety
Ciba-Geigy 1971	maternal weight loss	G	R		100 mg/kg	500 mg/kg	terat. study "minimum"
Ciba-Geigy 1984	dec. maternal wt gain; dec. food cons.	G	W	0, 1, 5, 75 mg/kg/day	1 mg/kg/day		terat. study "unacceptable", "ungradeable"
Woodard Research lab 1964	dec. body wt., food cons., F hemoglobin, hematocrit	F	D	0, 15, 150, 1500 ppm 2 years	150 ppm	1500 ppm	"supplementary"
American Biogenics Corp., 1986	dec. body wt. gain	F	F	R 0, 10, 70, 500, 1000 ppm	70 ppm		
Binns and Johnson 1970		?	S	maternal exposure throughout gestation and 1st 30 days of nursing	>15 mg/kg/day <30 mg/kg/day		

BROMACIL

<u>Source</u>	<u>Effects</u>	<u>Rt</u>	<u>Sp</u>	<u>Dose-schedule-duration</u>	<u>NOEL</u>	<u>LOEL</u>	<u>Notes</u>
EPA 1984d	dec. weight gain	F	D	2 years	250 ppm 6.25 mg/kg/day		
Hazleton 1966 #201-163	maternal toxicity	?	W	0, 50, 250 ppm	250 ppm (HDT)		teratology study "minimum"
EI Dupont (no date)	dec. growth; low RBC; inc. thyroid activity; enlargement of liver centolobular cells (all at 5000 ppm)	F	R	0, 50, 500, 2500/5000 ppm 80% WP for 90 days	500 ppm	2500 ppm	"minimum"
EI Dupont	dec. growth	F	R	0.005, 0.025, 0.125% for 2 years	250 ppm (0.025%)	0.125%	"minimum"
EI Dupont	dec. wt. gain	F	D	0.005, 0.025, 0.125% of 80% bromacil for 2 years	1250 ppm		"minimum"
Haskel Labs 1980 #893-80	testicular abnormalities inc. liver wt. at 5000 ppm	F	M	0, 250, 1250, 5000 ppm for 2 years	0.025%	0.125%	"acceptable"
					<250 ppm (LDT)		"minimum"

2,4-D

SOURCE	EFFECT(S)	RT	SP	DOSE-SCHEDULE-DURATION	# TESTED	# AFFECTED	NOEL	LOEL	NOTES
Drill and Hiratzka 1953	bleeding gums buccal mucosa necrosis stiff hind limbs liver/kidney alts. dec. lymphocyte counts	O/P	D	0.2, 5, 10, 20 mg/kg/day 5 days per w for 13 w			10 mg/kg/ day		
Hansen et al 1971		F	D	0, 10, 50, 100 or 500 ppm for 2 yrs	3 female, 3 males per group		500 ppm or 20 mg/kg/day		
EPA 1984	Histo changes in renal Tubules	F	M	0, 5, 15, 45, 90 mg/kg/day for 90 days			None est.		
EPA 1984	Histo changes in renal cortical tubules and inc. thyroid wt. at 1 mg/kg/day	F	R	0, 1, 5, 15, 45 mg/kg/day for 90 days			None est.		
Kociba et al 1949	mineralized deposits in, renal pelvis inc excret of coproporphyrin	F	R	3-30 mg/kg/day - 2 yrs			3 mg/kg day	10 mg/kg/ day	
Whitehead 1973	Food cons. & growth	F	Ch	10, 50, 100 ppm for 8 wks			(5ppm)	(10ppm)	chicks
Hazleton labs (1986)	Kidney effects	F	R	1-45 mg/kg/day 10 for wks			1 mg/kg/ day	5 mg/kg/ day	
Chem et al 1981	Kidney histo changes	?	R	13 weeks			15 mg/kg/ day		
Whitehead and Pettigrew 1972	Kidney enlargement at 5000 mg/kg	F	Ch	1000mg/kg (142 mg/kg bw) or 5000 mg/kg for 3 wks			1000 mg/kg		adults

2.4-D

SOURCE	EFFECT(S)	RT	SP	DOSE-SCHEDULE-DURATION	# TESTED	# AFFECTED	NOEL	LOEL	NOTES
(WHO 1984) Fabacher & Chambers 1974 Gambusia. Affins. Environ. Lett. 7:15-20 Meehan et al 1974 J.Fisher Res.Board Can. 31:480-485 King and Penfound 1946 Ecology 27:327-374 Ehiteneva & Chesnokova 1973 1973 Eksp. Vod. Toksikol. 4:56-67 (Russian)			Poikilo- Therms				1 mg/l water		2,4-D esters

2. 4-DP

<u>Source</u>	<u>Effects</u>	<u>Rt</u>	<u>Sp</u>	<u>Dose-schedule-duration</u>	<u>NOEL</u>	<u>LOEL</u>	<u>Notes</u>
EPA 1984f	blood enz. effects inc. liver/kidney wts.	F	R	90 days	5 mg/kg/day		
EPA 1984f	inc. liver wt.	F	M	18 months	100 mg/kg/day		
EPA 1984f	dec. wt. gain, hematocrit RBC count; prostatitis; kidney degeneration	F	R	2 years	50 mg/kg/day		
Hazleton labs no date	ataxia; dec. food intake	?	W	0, 25, 100 mg/kg	25 mg/kg (LDT)	100 mg/kg (HDT)	teratology study "minimum"
Litton Bionetics #7286 2-8-73		?	R	0, 10, 30, 100 mg/kg	>100 mg/kg		teratology study "minimum"
Huntington Research center #1-361	dec. body wt.	F	R	0, 125, 500, 1000, 2000 ppm	1000 ppm	2000 ppm	3 generation "minimum"
Central Inst. Vor Voedingsonderzoek (TNO) #R5419/a; 12/77	dec. blood Na, PCV; inc. liver/kidney wt.	F	R	0, 100, 500, 2500 ppm for 90 days	5 mg/kg (as reported)	25 mg/kg	"guideline"
TNO #R5555 6/78		F	D	0, 8, 20, 32 mg/kg for 4 wks	<8 mg/kg		"supplementary"
CDC research #CDC-AM-002-77 12-14-79	inc. bile retention liver wt., regeneration, degeneration	F	M	0, 25, 100, 300 mg/kg for 18 months	100 mg/kg	300 mg/kg	oncology study "guideline"
CDC research #CDC-AM-001-77 4-18-80	dec. wt. gain, hematocrit, RBC count; renal degen., chronic prostatitis; testicular atrophy	F	R	0, 25, 50, 200/150 mg/kg for 2 years	50 mg/kg	150 mg/kg	oncology study "guideline"
Inst. Env. Tox., Japan no date	dec. urinary specific gravity and/or protein	F	R	0, 100, 300, 1000, 3000 ppm for 2 years	100 ppm	300 ppm	oncology study "guideline"
Inst. Env. Tox., Japan 12-14-81	dec. hematocrit, hemo- globin, A/G ratio; inc. in Alk. phosphatase, total bilirubin, albumin	F	R	100, 300, 1000, 3000 ppm for 90 days	300 ppm	1000 ppm	"supplementary"

DALAPON

<u>Source</u>	<u>Effects</u>	<u>Rt</u>	<u>Sp</u>	<u>Dose-schedule-duration</u>	<u>NOEL</u>	<u>LOEL</u>	<u>Notes</u>
Paynter et al 1960 in USDA 1984	inc. kidney wts.	F	R	2 years	15 mg/kg/day		
Paynter et al 1960 in USDA 1984	inc. kidney wts.	F	D	52 weeks	50 mg/kg/day		no abnormal
Paynter et al 1960	inc. kidney wts.; dec. growth; slight changes in kidney, liver histology	F	R	0, 10, 30, 100, 300, or 1000 mg/kg/day for 97 days	100 mg/kg/day (male) 10 mg/kg/day (female)		pathology/histol.
Thompson et al 1971 Kenaga et al 1974	maternal toxicity	G	R	0, 250, 500, 1000, 1500 or 2000 mg/kg/day days 6-15 of gestation	500 mg/kg/day		teratology study
Dow Chemical 1983	inc. liver wt.	F	D	0, 2, 60 or 200 mg/kg/day for 2 years	60 mg/kg/day		
Hazleton labs 1956		F	R	0, 0.01 or 0.03% in diet for 2 years	not established		"unacceptable" according to Cal.
Hazleton labs 1956		OC	D	0, 15, 50, 100 mg/kg for 52 weeks	not established		"unacceptable" according to Cal.

DICAMBA

Source	Effects	Rt	Sp	Dose-schedule-duration	NOEL	LOEL	Notes
EPA 1985d		?	R		250 mg/kg/day		EPA "in house value"
EPA 1985d	slight liver cell alts.	?	R	90 days	500 ppm/25 mg/kg/day		
IRDC #163-436 9/77	maternal toxicity (mortality; dec. wt. gain)	?	W	0, .5, 1, 3, 10, or 20 mg/kg/day	10 mg/kg/day	20 mg/kg/day	teratology study "supplemental"
IRDC #163-436 10/78	dec. weight gain	?	W	0, 1, 3, or 10 mg/kg/day	3 mg/kg/day	10 mg/kg/day	"supplementary" teratology study
Toxigenics #450-0460	dec. food cons.; wt loss	G	R	0, 64, 160, 400 mg/kg days 6-19 of gestation	160 mg/kg/day	400 mg/kg/day	"minimum" teratology study
U of Cincinnati 1962	dec. food cons; wt. loss	F	D	0, 5, 25, 50 ppm in food for 2 years	5 ppm (male body wt.) 25 ppm (female body wt.) 50 ppm (hematology, histopathology, urinalysis, organ wts.)		"supplementary" chronic study
U of Illinois 1962	slight liver cell necrosis and cytoplasmic vacuolization	F	R		500 ppm	800 ppm	dimethylamine salt of Banvel
Edson and Sanderson 1965	Inc. liver weight	F	R	0 to 3162 ppm	316 ppm	1000 ppm	

DIURON

<u>Source</u>	<u>Effects</u>	<u>Rt</u>	<u>Sp</u>	<u>Dose-schedule-duration</u>	<u>NOEL</u>	<u>LOEL</u>	<u>Notes</u>
EPA 1984i		F	R	2 years	125 ppm or 6.25 mg/kg/day		
EPA 1984i		F	D		25 ppm or .625 mg/kg/day		
Hodge et al 1967	dec. growth; slight anemia; abnormal pigments; hemosiderosis	F	R	2 years	250 ppm		
Hodge et al 1967		F	D	2 years	250 ppm		
EPA memorandum; Diuron 8-20-82		F	R	0, 25, 125, 250, 2500 ppm for 2 years	25 ppm		no core grade
EPA memorandum; Diuron 8-20-82	wt. loss; dec. RBC counts; inc. liver wt.; inc. liver cell pigmentation	F	D	0, 25, 125, 250, 1250 ppm for 2 years	25 ppm		no core grade
Khera et al 1979	dec. maternal body wt.	G	R	0, 125, 250, 500 mg/kg/day days 6-15 of gestation	250 mg/kg/day		"supplemental" teratology study

FOSAMINE

<u>Source</u>	<u>Effects</u>	<u>Rt</u>	<u>Sp</u>	<u>Dose-schedule-duration</u>	<u>NOEL</u>	<u>LOEL</u>	<u>Notes</u>
Schneider and Kaplan 1983 in USDA 1984	inc. stomach wt.	F	D		100 ppm 2.5 mg/kg/day		
Schneider and Kaplan 1983 in USDA 1984		F	R		5000-10,000 ppm 250-500 mg/kg/day (HDT)		
Schneider and Kaplan 1983 in USDA 1984a	none	F	R	0, 200, 1000, 5000/10,000 ppm for 90 days	5000-10,000 ppm		
Schneider and Kaplan 1983 in USDA 1984a	inc. heart, stomach wts.	F	D	0, 200, 1000, 5000, 7000 ppm for 6 months	1000 ppm or 40 mg/kg/day (calc used by Crump)		worst case NOEL

GLYPHOSATE

Source	Effects	Rt	Sp	Dose-schedule-duration	NOEL	LOEL	Notes
EPA 1984k		F	R	26 months	30 mg/kg/day		
Monsanto: 1982c in 1984a	"systemic"	D	W	0, 100, 1000, 5000 mg/kg/day for "15 out of 21 days"	5000 mg/kg/day		IDRC NOEL 1000, LOEL 5000
Monsanto: 1972 in 1984a	dec. food cons., body wt.; inc. mortality	D	W	32% aq. solution by Vol. (5 X intended conc.)	3 x 6.4% by Vol.		
Monsanto: 1983a in 1984a		I	R	.36 mg aq solution/ 1 air 6 hrs/day, 5 days/wk, 30 days			
Monsanto: 1979h in 1984a	inc. in relative and absolute lung wt.	F	R	0, 200, 2000, 5000, 12500 ppm or 0, 13.5, 135, 340, 820 mg/kg/day, 90 days	2000 ppm or 135 mg/kg/day		
Monsanto: 1979i in 1984a	dec. growth at 50,000 ppm	F	M	up to 50,000 ppm for 90 days	10000 ppm or 2305 mg/kg/day		
Monsanto #83-137 8-22-85	apparent dec. in relative and absolute pituitary wts.	O	D	0, 20, 100, 500 mg/kg/day for 1 year	20 mg/kg/day *	100 mg/kg/day	tentative NOEL LOEL, EPA reqs. more data
Monsanto, 1985 #ML-83-137		O	D	96% glyphosate in capsul 0, 20, 100, 500 mg/kg/day for 1 year	>500 mg/kg/day *		
Monsanto, 1983 #77-2061	central lobular hepatic necrosis/hypertrophy; chronic interstitial nephritis; proximal tubule epithelial basophilia and hypertrophy	F	M	0, 1000, 5000, 30000 ppm 99.7% glyphosate (for 2 years?)	5000 ppm		chronic feeding study
IRDC #IR-79-018 2-29-80	inc. mortality; misc. clinical signs	G	W	0, 75, 175, 350 mg/kg/day 98.7% glyphosate, days 6-27 of gestation	175 mg/kg/day (maternal systemic)		teratology study EPA "minimum" Cal "acceptable"
Biodynamics #77-2062 9-18-81	inc. mortality; dec. wt. gain; clinical pathology	F	R	0, 3, 10, 31 mg/kg/day for 26 months	>31 mg/kg/day systemic		onco study "minimum"
Biodynamics #77-20663 7-6-82		?	R	3 generations	10 mg/kg/day		"minimum"
IRDC #401-054 3-21-80	maternal tox.; inc. mortality; dec. wt. gain; alt. gen. appearance	?	R	0, 300, 1000, 3500 mg/kg/day	1000 mg/kg/day maternal tox.	3500 mg/kg/day	teratology study "minimum"

* California 1 liners (12-2-86) does not acknowledge EPA's summary (Guidance for the Registration of Pesticides Containing Glyphosate, 6-30-86) that states a "tentative" NOEL of 20 mg/kg/day based upon "apparent" wts in pituitary weight at 100 and 500 mg/kg/day. No evaluation of "tentative" or "apparent" available. The Cal. 1 liner does, however, claim "no data gap" in their review of its chron study.

HEXAZINONE

<u>Source</u>	<u>Effects</u>	<u>Rt</u>	<u>Sp</u>	<u>Dose-schedule-duration</u>	<u>NOEL</u>	<u>LOEL</u>	<u>Notes</u>
EPA 1984i	inc. liver wt., liver cell size	F	R	2 years	10 mg/kg/day		
		F	M	2 years	30 mg/kg/day		
Haskel labs (Dupont) 1973	dec weight gain	F	R	0, 200, 1000, 5000 ppm 3 months	1000 ppm	5000 ppm	
	dec. wt. gain, inc. alkaline phosphatase; dec albumin: globulin ratio	F	D	0, 200, 1000, 5000 ppm 3 months	1000 ppm	5000 ppm	"minimum"
Haskel labs 1977	dec. wt. gain	F	R	0, 200, 1000, 2500 ppm 2 years	200 ppm	1000 ppm	"minimum"
IRDC #125-026 6-23-81	liver hypertrophy, hyperplastic nodules focal necrosis	F	M	0, 200, 2500, 10,000 ppm 2 years	200 ppm (30 mg/kg/day)	2500 ppm	may be same as LA EPA 1984i

PICLORAM

<u>Source</u>	<u>Effects</u>	<u>Rt</u>	<u>Sp</u>	<u>Dose-schedule-duration</u>	<u>NOEL</u>	<u>LOEL</u>	<u>Notes</u>
BarnaLloyd et al., 1982 in Mullison, 1985	inc. liver wts.	F	D	0, 7, 35, 175 mg/kg/day	7 mg/kg/day	35 mg/kg/day	
Lynn 1965	liver histo., necrosis bile duct prolif.; alterations in: blood chem, body/organ wts., mortality	F	R	75, 225, 750 mg/kg/day or 1000, 3000, 10,000 ppm in diet for 90 days	10,000 ppm or 75 mg/kg/day (conversions reported by author)		
NCI 1978		F	R	1250 to 20,000 ppm for 6 wks	10,000 ppm		
NCI 1978		F	M	1250 to 30,000 ppm for 6 wks	5000 ppm		
Dow chemical 1984 in USDA 1984a	inc. relative, absolute liver wts. at 150 mg/kg and kidney wts. at 300 mg/kg	F	R	0, 15, 50, 150, 300, 500 mg/kg/day for 13 wks	50 mg/kg/day		
Dow chemical 1984 in USDA 1984a	unspecified toxic liver and gastric mucosa effects at 3000 mg/kg/day	F	M	0, 30, 100, 650, 1000, 3000 mg/kg/day for 32 days	1000 mg/kg/day		
Dow chemical 1984 in USDA 1984a	inc. relative, absolute liver wt	F	D	0, 7, 35, 175 mg/kg/day for 6 months	7 mg/kg/day		worst case NOEL
Dow chemical 1986	inc. size of hepatocytes, functional properties of liver	O	R	0, 20, 60, 200 "mg/kg in the diet for 2 years" (97% picloram, 197 ppm hexachlorobenzene)	20 mg/kg	60 mg/kg	"acceptable"

TEBUTHIURON

<u>Source</u>	<u>Effects</u>	<u>Rt</u>	<u>Sp</u>	<u>Dose-schedule-duration</u>	<u>NOEL</u>	<u>LOEL</u>	<u>Notes</u>
EPA 1984o		F	M	119 day	554 ppm 83.1 mg/kg/day		
EPA 1984o	inc. thyroid/body wt. ratios; inc in blood enzyme levels	F	D	3 month	500 ppm 12.5 mg/kg/day		
Elli Lilly Res. lab #s R03780 and R08780 11-81		?	R	2 generations	>100 ppm		"supplementary"
Elli Lilly Res. lab 4-75		?	R	3 generations	800 ppm (HDT)		
Elli Lilly Res. lab 1972	dec. growth; pancreatic lesions	F	R	90 days	1000 ppm	2500 ppm	
Elli Lilly Res. lab 1972	inc. thyroid, spleen wts.	F	D	90 days	12.5 mg/kg	25 mg/kg	
Elli Lilly Res. lab 1976	dec. growth	F	Cw	162 days	30 ppm	100 ppm	
Elli Lilly Res. lab 1972	dec. growth	F	Ch	30 days	1000 ppm	2500 ppm	
Elli Lilly Res. lab 1976	dec. growth	F	R	2 years	400 ppm 20 mg/kg	800 ppm 40 mg/kg	

SIMAZINE

<u>Source</u>	<u>Effects</u>	<u>Rt</u>	<u>Sp</u>	<u>Dose-schedule-duration</u>	<u>NOEL</u>	<u>LOEL</u>	<u>Notes</u>
EPA 1984n		F	R	2 years	> 100 ppm or 5 mg/kg/day		
EPA 1984n		F	D	2 years	1500 mg/kg/day (HDT)		
Woodward lab 1965		F	M	20 days	250 mg/kg/day (HDT)		
Ciba-Geigy #62-83 5-17-86	dec. food cons., wt. gain abortions	G	W	0, 5, 75, 200 mg/kg	5 mg/kg	75 mg/kg	teratology study "guideline"
Ciba-Geigy #85018 4-10-85	dec. RBC, WBC counts inc. levels of cholesterol, inorganic phosphate	F	R	3 weeks	<200 ppm (LDT)		"supplementary"
Ciba-Geigy #85022 4-12-85	dec. albumin; increased: globulin, ketone levels; urinary specific gravity	F	D	3 weeks	200 ppm	2000 ppm	"minimum"

TRICLOPYR

Source	Effects	Rt	Sp	Dose-schedule-duration	NOEL	LOEL	Notes
EPA 1984p, USDA 1984		F	R, M	2 years	30 mg/kg/day (HDT)		
Dow chemical, in USDA 1984	dec. food cons, wt. gain	F	D	228 days	<5 mg/kg/day		
40 CFR part 180 5(84) 18485 5-1-85	effects not representative of human effects	F	D	6 months	2.5 mg/kg (per day?; HDT)		
Humiston et al 1975	dec. growth; dec. liver wt. inc. brain, kidney wts.	F	R	90 days	30, 100 mg/kg/day (male, female) (HDT)		
EPA 1984	dec. wt. gain at 200, 300 mg/kg/day for females and 100 mg/kg only for males	F	R	0, 30, 100, 200, 300 mg/kg/day, 14 days	30 mg/kg/day		
Dow chemical, in USDA 1984a	dec. liver wt. in males at 60 mg/kg/day	F	M	6, 20, 60 mg/kg/day for 90 days	20 mg/kg/day (males) 60 mg/kg/day (females)		
Mollelo et al 1976		GI	Mk	28 days	30 mg/kg/day (HDT)		
Quast et al 1976		F	D	0, 5, 10, 20 mg/kg/day for 228 days	none established	5 mg/kg/day	
Quast et al 1977	dec. excretion of phenosulfonphthalein	F	D	0, 0.1, 0.5, 2.5 mg/kg/day for 183 days	0.5 mg/kg/day	2.5 mg/kg/day	worst case NOEL EPA "minimum" Cal "unacceptable"
Liton Bionetics #2538 11-4-86		F	R	0, 3, 10, 30 mg/kg 3 generations	>30 mg/kg/day (HDT)		reproduction study "minimum"
Smith et al 1977	maternal mortality	G	W	0, 25, 50, 100 mg/kg/day days 6-18 of gestation	not established	25 mg/kg/day (LDT)	teratology study
Dunn et al 1980 (IBT; EPA acceptable)		F	R	3, 10, 30 mg/kg/day for 2 years	30 mg/kg/day (HDT)		oncology study
Mollelo et al 1979	no effect (wt. gain; organ wt. ratios; hematology; urinalysis; clinical chem.; histology)	F	M	0, 3, 10, 30 mg/kg/day for 2 years	30 mg/kg/day (HDT)		oncology study

2.4-D-D

SOURCE	EFFECT(S)	RT. SP	DOSE-SCHEDULE-DURATION	# TESTED	# AFFECTED	NOEL	LOEL	NOTES
EPA 1984e		F D	0-500 ppm or 0-12.5 mg/kg for 2 yrs			12.5 mg/kg/day		
EPA 1984e		F R	1250 ppm or 62.5 mg/kg/day for 2 yrs			62.5 mg/kg/day		
EPA 1985c	Kidney effects	F R				1 mg/kg/ day	5 mg/kg/ day	
Kay et al. 1965		D W	.636% 7 or 3.13% 7 hrs/day x 5 days/wk for 3wks			none spec		
Bucher 1946		G M	up to 93 mg/kg/day 3 wks to 3 months			93 mg/kg/ day		
Bucher 1946	dec. growth	SQ M	50 to 90 mg/kg once or twice daily 3 wks to 3 mos			70 mg/kg/ day	70 mg/kg/ day	
Rowe and Hymas 1954	GI irritation dec. growth rate cloudy swelling of liver	GI R	0,3,10,30,100,300 mg/kg/day 5 times/w for 4 weeks			30 mg/kg/ day	100 mg/kg/ day	
Hill and Carlisle 1947		F R	0, 100, 200 or 400 ppm for 30 days	7 rats/dose				No effect at 1000 ppm in food for 1 month, IARC 15, 1977
Bjorklund and Erne 1966		F R	1000 ppm for 10 months					no detrimental effects occurred
Rowe and Hymas 1954	dec. Growth rate inc. Mortality inc. liver wt Cloudy swelling of liver (1000 mg/kg)	F R	0,100,300,1000,3000 10k ppm mg/kg/ diet for 113 days			300 ppm 15 mg/kg/ day	1000 ppm	
Hansen et al. 1971	growth & organ wt., hematology, survival	F R	0, 5, 25, 125 or 1250 ppm for 2 yrs			1250 ppm or 62.5 mg/kg/day		

RESOURCES

- (Crump) Worst-case analysis study on Forest Plantation Herbicide use
C. S. Crump and Co., Inc. 1201 Gains St., Ruston, LA 71270
- (LAI) Labatt-Anderson
Supplement to the Western Oregon Program-Management of Competing Vegetation.
Draft Environmental Impact Statement U. S. Dept. of Interior, Bureau of Land Management
- (Hayes) Hayes, Wayland J. Pesticides Studied in Man: Chap 11 Herbicides
Williams and Wilkins. Baltimore, MD 1982
- (IARC) IARC Monographs on the Evaluation of the Carcinogenic risk of Chemicals to man.
Some fumigants, the Herbicides 2,9-D and 2,4,7-T, Chlorinated Dihencodioxins and Miscellaneous
Industrial Chemicals Volume 15 International Agency for Research on Cancer Aug 1977
- (Cal one liners) California Dept. of Food and Agriculture Medical Toxicology Branch
Summary of Toxicology Data: 2,4-D . (One liners)
- (WHO 1984) Environmental Health Criteria 29. 2,4-Dichlorophenoxyacetic acid (2,4-D)
World Health Organization, Geneva 1984

LEGEND

RT = route: D- dermal; F- in food; G- gavage; GI- gastric intubation; I- inhalation; OC- oral capsule; O- oral, not further described;
O/P- oral pellet; SQ- subcutaneous; ?- not stated

SP = species: Ch - chicken; Cw- cow; D- dog; H- hamster; M- mouse; Mk- monkey; R- rat; S- sheep; W- rabbit

LDT = lowest dose tested (when indicated)

HDT = highest dose tested (when indicated)

ppm - For dosages in laboratory animals expressed as concentration in the diet, i.e. in parts per million (ppm), approximate conversions to mg/kg/day can be made with the following factors: rat, 1 ppm = 0.05 mg/kg/day; mouse, 1 ppm = 0.150 mg/kg/day; rabbit, 1 ppm = 0.030 mg/kg/day; dog, 1 ppm = 0.025 mg/kg/day

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Appendix H
Human Health Risk
Assessment
(Qualitative)

Section 3
Data for Analysis
of Mutagenic
and Carcinogenic
Toxicity

DATA FOR ANALYSIS OF MUTAGENIC AND CARCINOGENIC TOXICITY

Footnotes

a) Data for each compound was evaluated for: 1) the quantity and quality of available studies, 2) the consistency of effects between studies, and 3) the strength of association and/or dose-relatedness of the effect, with a consideration for the potency of the chemical in producing the effect.

I (Inadequate) = data are so limiting and / or of poor quality so as to preclude any meaningful judgement of the potential for the chemical to produce the effect in question. Further studies are necessary. The summary result of available data is shown in brackets to denote the tenuous nature of the findings.

M (Minimal) = sufficient data are available to make a *cautionary* judgement about the ability of the chemical to produce the effect in question. Further testing is desirable.

A (Adequate) = data are of sufficient abundance and quality to make a reasonably confident judgement about the ability of the chemical to produce the effect in question. Further testing would be of limited value, and is not essential.

- = effect is consistently negative or insignificant,

+ = effect is consistently positive

± = effect is equivocal, or inconsistently positive / negative.

* = no studies are available to judge

b) Although almost all of the numerous acceptable mutagenicity / cytogenicity studies on amitrole have been negative, at least 4 four cell transformation studies have tested positive, suggesting that amitrole may have cancer promoting activity.

c) Although numerous different mutagenicity studies have been performed on atrazine, most have been published only as abstracts or summary reports, so detailed evaluations of quality are not possible . One reasonably consistent theme that emerges, however is that atrazine appears to be activated to mutagenic metabolite(s) by many plant activating systems, but generally not by mammalian activating systems. There is limited evidence suggesting that atrazine is positive in cell transformation assays, but only at quite high concentrations *in vitro* .

Review Table for Mutagenicity of Amitrole

<u>TEST ORGANISM TYPE</u>	<u>Response</u>	<u>Quality</u> ¹	<u>Reference</u>
I. Point Mutations or Specific Locus tests			
Microbial assays:			
1. Ames TA98,100,1535, 1537, 1538, \pm S9	-	A	Moriya et al., 1983
2. Ames TA98,100,1535, 1537, 1538, + S9	-	A	Falck et al., 1985
3. Ames TA 1535, 1536, 1537, 1538 \pm S9	-	A	Carere et al., 1978
4. <i>S. coelicolor</i> A3(2), <i>his</i> A ⁻ 1	\pm	?	"
5. <i>S. cerevisiae</i> D3 (recombination), S9	-	A	Simmon, 1979
6. <i>Aspergillus nidulans</i> , 8-azaguanine resist.	-	?	Bignami et al., 1977
7. " " , somatic recombinations	\pm	?	"
8. " " , non-dysjunction	\pm	?	"
9. " " , non-dysjunction	+	A	Morpurgo et al., 1979
10. Ames test plus nitrate	+	A	Braun, 1977
11. Ames test	+	?	Venitt & Crofton-Sleigh, 1981
12. "Bacterial forward and reverse mutation" 7 additional studies negative, 1 positive	7-, 1+	R	NHMRC, 1984 ³
13. Yeast reverse mutation, mitotic gene conversion & non-dysjunction, 4 additional studies	3-, 1+	R	"
14. Ames test, "49 tests in different strains"	-	R	EPA, 1985 ²
Drosophilla tests:			
1. mutation	-	A	Laamanen et al., 1977
2. non-dysjunction	-	A	"
3. recessive lethal test	-	A	"
4. non-dysjunction	-	I (abs)	Sorsa & Gripenberg, 1976
mammalian cell cultures:			
1.			
in vivo host mediated assays			
1. <i>S. typhimurium</i> TA 1530, 1538	+	A	Simmon et al., 1979
2. <i>S. cerevisiae</i> D3	+	A	"
II. DNA Damage / Repair Tests			
Microbial assays:			
1. <i>E. coli</i> <i>pol</i> A reversion	-	?	Bamford et al., 1976
2. <i>E. coli</i> <i>pol</i> A reversion, \pm S9	-	A	Rosenkranz & Poirer, 1979
3. <i>E. coli</i> WP2/WP100 <i>rec</i> (<i>uvr</i> A ⁻ <i>rec</i> A ⁻)	-	A	Mamber, et al., 1983
4. <i>E. coli</i> DNA DNA-cell binding assay \pm S9	+	I	Kubinski et al., 1981
5. "microbial DNA repair", 2 addit. studies reviewed	-	R	NHMRC, 1984 ³
6. <i>E. coli</i> WP2 <i>her</i> , \pm S9	-	A	Moriya et al., 1983
7. "SOS Chromtest" induction of <i>sfi</i> A gene in <i>E. coli</i>	-	?	Quillardet et al., 1985
8. <i>E. coli</i> WP2 <i>uvr</i> A	-	A	Falck et al., 1985

Mammalian Cell culture:

1. unscheduled DNA synthesis	+	I (abs)	Begnini & Dogliotti, 198
2. UDS in HeLa cells, \pm S9	+S9	?	Martin & McDermid, 198 in EPA, 1985 ²

Other:

1. <i>in vivo</i> inhibition of testicular DNA synthesis	-	A	Seiler, 1977
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III. Chromosomal Abberations / Cytogenetic tests

Drosophila tests:

1. recessive lethal assay	-	A	Vogel et al., 1980
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mammalian cell cultures:

1. sister chromatid exchange, CHO cells	-	R	EPA, 1985 ²
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in vivo animal studies:

1. sperm head abnormalities, mice	-	A	Topham, 1980
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in vivo / *in vitro* human studies:

1. aneuploidy / aberrations	-	?	Meretoja et al., 1976
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plant material assays:

1. <i>Pelargonium zonale</i> , chlorophyll defects	+++	I (abs)	Polheim et al., 1976
2. "mutations & chromosomal effects	-	R	NHMRC, 1984 ³

IV. Cell Transformation Assays

Mammalian cell cultures:

1. human lymphoblast transformation	+	?	Meretoja et al., 1976
2. 4 <i>in vitro</i> transformation studies, rat & hamster	4+	R	cited in EPA, 1985 ²
3. Syrian Hamster embryo, HGPRT and TAPase loci	+	A	Tsutsui et al., 1984

FOOTNOTES:

1.

1. Quality assessment is a judgement of the overall validity of the study. A = acceptable (this indicates that useful information is provided), I = inadequate (this indicates flaws in study design, data interpretation, or questionable significance), ? = unable to judge (when uncommon study is used with little basis for comparison or no reference compounds, but apparently acceptable study design and interpretation. May also be used when there is inadequate information to make a judgement), R = signifies that the study was cited in a review, but original study was not evaluated. If the review (e.g. EPA or CDFA) gave a quality rating, this is indicated prior to the R.

2 U.S.E.P.A.: Amitrole Position Document 2/3, dated 9/30/85. Mutagenicity summary:

" Amitrole has been evaluated in a variety of mutagenicity test systems. Although positive results were reported [in 2 studies], 49 other *Salmonella* gene mutation tests and 9 other *E. coli* tests were negative. The validity of the two positive studies is questionable. The weakly positive results by Carere *et al.* were in an unvalidated system using unusual bacteria. The mechanisms for this [*sic*] positive results

reported for the DNA repair assays can not be determined without positive gene mutation or chromosome aberration assays. The negative results in the sister chromatid exchange assay in mammalian cells in culture (which is a very sensitive assay) and the chromosome aberration assay in culture human lymphocytes or *in vivo* mouse bone assays. [sic] Amitrole does not present a potential for heritable genetic effects.

Amitrole induces transformation in cultured cells and was positive in four *in vitro* transformation studies using rat and hamster cells ... following treatment of 0.1 to 100 µg/ml. This test is used to establish the malignant activities of test compounds on mammalian cells *in vitro*. Cells treated *in vitro* with chemical carcinogens give rise to foci of cellular growth superimposed on the cell monolayer. If these foci are picked from the cultures, grown to larger numbers, and injected into animals, a malignant tumor will be obtained, in most cases. Therefore, the appearance of piled-up colonies in treated cell cultures is correlated with malignant transformation. In addition, weak cellular transformation capacity was observed in EUE cells (no data presented, only summary) (Benigni, 1980.)

The Agency concludes that "available transformation assays neither determined a mechanism for tumor formation nor necessarily demonstrated that a transformation inducer is genotoxic. These results support oncogenicity potential but not necessarily mutagenicity potential"

3. National Health and Medical Research Council [of Canada]: Report of the Working Party on Amitrole, ninety-seventh session, June 1984. Mutagenicity summary:

"Although sporadic positive responses have been recorded in various microbial assays as well as in assays for aneuploidy in yeast, and for transformation, sister chromatid exchanges and unscheduled DNA synthesis in mammalian cells, these reports suffer certain statistical or technical deficiencies and are not considered to provide adequate evidence for amitrole as a DNA-damaging, mutagenic or transforming chemical". This report is a review of 201 references related to amitrole mutagenicity and carcinogenicity.

Because of the large number of studies available on the mutagenicity of amitrole, and because these studies have been thoroughly reviewed previously by qualified agencies (see above), this review table is not exhaustive, but represents an independent review of many of the most useful mutagenicity studies available.

Review Table for Mutagenicity of Asulam

<u>TEST ORGANISM TYPE</u>	<u>Response</u>	<u>Quality</u> ¹	<u>Reference</u>
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I. Point Mutations or Specific Locus tests

Microbial assays:

1. Ames TA 98, 100, 1535, 1537, 1538, \pm S9	--	IR ² AR ³	CDFA, 1986; EPA, 1986;
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mammalian cell cultures:

1.

in vivo host mediated assays:

1.

II. DNA Damage / Repair Tests

Microbial assays:

1.

Mammalian Cell culture:

1. UDS in HeLa S3 cells	-	IR ³	CDFA, 1986
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III. Chromosomal Abberations / Cytogenetic tests

drosophila tests:

1.

mammalian cell cultures:

1.

in vivo animal studies:

1. dominant lethal test, mice	-	IR ² ?R ³	CDFA, 1986; EPA, 1986;
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in vivo / *in vitro* Human studies:

1. PHA-M stimulated human lymphocytes	-	IR ³	CDFA, 1986
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plant material assays:

1.

IV. Cell Transformation Assays

1. C3H/10T ^{1/2} cell transformation	-	IR ² AR ³	CDFA, 1986; EPA, 1986;
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Footnotes:

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2. CDFA, California Department of Food and Agriculture, Medical Toxicology Branch, Summary of Toxicology Data- Asulam, SB#950-219, Tolerance # 360, dated December 4, 1986.

3. EPA Tox one liners, No.62A, Asulam, dated 1/29/85

Review Table for Mutagenicity of Atrazine

<u>TEST ORGANISM TYPE</u>	<u>Response</u>	<u>Quality</u> ¹	<u>Reference</u>
I. Point Mutations or Specific Locus tests			
Microbial assays:			
1. Ames TA98, 100, 1535,1537, ±S9 (SRI)	-	IR ² AR ³	CDFA, 1986 ² , EPA 1986 ³
2. Ames TA 100, 1535, 1537, ± S9 (Ciba-Geigy)	-	IR ²	CDFA, 1986
3. Ames, old nomenclature, -S9	-	I	Andersen et al., 1972
4. Reversion of bacteriophages N17 and AP72 to T ₄	-	I	" " "
5. Ames TA98, 100, 1535,1537, ±S9 (SRI)	-	A	Eisenbeis et al., 1981
6. <i>S. cerevisiae</i> , ± mouse S9	-	I(abs)	deBertoldi et al., 1980
7. <i>Aspergillus nidulans</i> , ± mouse S9	-	I(abs)	deBertoldi et al., 1980
8. Ames test, "9 strains"	-	R ⁴	Loprieno & Adler, 1980
9. <i>S. typhimuriam</i> , 8-azaguanine resistance	-	R ⁴	Loprieno & Adler, 1980
10. <i>S. coelicolor</i> , strp resistance (with potato microso)	+	R ⁴	Loprieno & Adler, 1980
11. <i>S. cerevisiae</i> , + potato microsomes	-	R ⁴	Loprieno & Adler, 1980
12. <i>E. coli</i> , ampicillin resist.	-	R ⁴	Loprieno & Adler, 1980
13. <i>Schizosaccharomyces pombe</i> , ± mouse S9, ± maize S9	-	I(abs)	Chollet et al., 1980
14. Ames, TA98, + <i>Z. maize</i> (plant) metabolic activation	+	A	Plewa et al., 1984
mammalian cell cutures:			
1. V79 cells, 6-thioguanine resist., + potato microsomes	+	R ⁴	Loprieno & Adler, 1980
<i>in vivo</i> host mediated assays:			
1. <i>Salmonella</i> TA 1535 or 1538, in mice	-	R ²	CDFA, 1986
2. <i>E. coli</i> forward mutation	-	I(abs)	deBertoldi et al., 1978
3. <i>E. coli</i> forward mutation	+	I(abs)	Solte and Neale, 1980
II. DNA Damage / Repair Tests			
Microbial assays:			
1.			
Mammalian Cell culture:			
1. UDS, EUE human cells, with potato microsomes	+	R ⁴	Loprieno & Adler,1980
III. Chromosomal Abberations / Cytogenetic tests			
drosophila tests:			
1. dominant lethals (egg hatching success)	+	I(abs)	Murnik, 1976
2. recessive lethal	-	I(abs)	Loprieno et al., 1980
mammalian cell cultures:			
1. chinese hamster ovary cells	-	R ⁴	Loprieno & Adler, 1980

2. mouse bone marrow cells	+	R	Ehling, 1980, in Crump et al., 1986
3. mouse bone marrow cells	-	R ⁴	Loprieno & Adler, 1980
4. sister chromatid exchange, hamster cells	-	R ⁴	Loprieno & Adler, 1980

in vivo animal studies:

1. mouse dominant lethal mutations	-	R	Ehling 1980, in Crump, 1986;
2. mouse dominant lethal mutations (spermatids)	+	R ⁴	Loprieno & Adler, 1980
3. mouse bone marrow, "chromosome analysis"	+	I(abs)	Kliesch & Adler, 1983
4. mouse bone marrow, micronucleus test	-	I(abs)	Kliesch & Adler, 1983
5. mouse bone marrow, "clastogenic activity"	-	I(abs)	Chollet et al., 1980
6. bone marrow cells, "spermatogonia and diakinesis"	-	I(abs)	Chollet et al., 1980
7. mouse dominant lethal, post-implantation loss	-	I(abs)	Chollet et al., 1980
8. mouse dominant lethal, pre-implantation loss	+	I(abs)	Chollet et al., 1980

in vivo / *in vitro* Human studies:

1. cultured human lymphocytes	-	I(abs)	Ghiazza et al., 1984
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plant material assays:

1. <i>Tradescantia</i> micronucleus test	-	I	Ma et al., 1984
2. <i>Pelargonium zonale</i> chlorophyll defects	-	I(abs)	Pohleim et al., 1976
3. <i>Vicia</i> root tip SCE and aberrations	+	R	Ma, 1982
4. Maize <i>wx</i> locus assay	+	A	Plewa et al., 1984

IV. Cell Transformation Assays

1. fibroblasts, SHEM cells (early passage), no S9	+	A	Dunkel et al., 1981
2. virus infected, R-MuLV-RE cells	+	A	Dunkel et al., 1981

Footnotes:

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2. California Department of Food and Agriculture, Medical Toxicology Branch, Summary of Toxicology Data - Atrazine SB950-008, Tolerance #220, dated August 11, 1986.

3. EPA one liner no. 63 - Atrazine, dated 07/09/85

4. This paper is a preliminary report of "a coordinated comparative test programme to assess the mutagenic effects of five chemical in as many different assay systems as possible." This study was sponsored by the European Economic Community. Few details of experimental protocols and design are provided, so no evaluation of adequacy of studies can be made.

Review Table for Mutagenicity of Bromacil

<u>TEST ORGANISM TYPE</u>	<u>Response</u>	<u>Quality</u> ¹	<u>Reference</u>
I. Point Mutations or Specific Locus tests			
Microbial assays:			
1. Ames test, TA100, 98, 1535, 1537, 1538 ± S9	-	A	Moriya et al., 1983
2. Ames test, TA 100, 1535, 1537, 1538, +mouse S9	-	R ²	Waters et al., 1981
4. <i>S. cerevisiae</i> D3 recombination	-	R ²	"
6. <i>S. cerevisiae</i> D3 and D7 assays, ±S9	-	I(abs)	Riccio et al., 1981
7. Ames test, strains & methods unspecified	+	I	Njagi & Gopalan 1980
9. bacteriophage AP72 of <i>E. coli</i>	-	I,R	McGahen and Hoffman, 1966, cited in CDFA, 1986 ³ .
mammalian cell cultures:			
1. mouse lymphoma L5178Y forward mutation for TK±	+	R	Waters et al., 1982, cited in CDFA, 1986 ³ .
<i>in vivo</i> host mediated assays:			
1.			
II. DNA Damage / Repair Tests			
Microbial assays:			
1. <i>E. coli</i> WP2 <i>hcr</i>	-	A	Moriya et al., 1983
2. <i>E. coli</i> WP2 <i>uvrA</i> ⁻	-	R ²	Waters et al., 1981
3. <i>E. coli</i> W3110 & P3478 repair deficient, (Pol A)	-	R ²	"
4. <i>B. subtilis</i> H17 and M45 <i>rec</i> ⁺	-	R ²	"
Mammalian Cell culture:			
1. UDS in human fetal lung fibroblasts (WI-38 cells)	-	R ²	Waters et al., 1981
III. Chromosomal Abberations / Cytogenetic tests			
<i>drosophila</i> tests:			
1. complete and/or partial loss of chromosomes <i>mus</i> - 302 repair defective females	-	A	Woodruff et al., 1983
2. sex-linked recessive lethal test	+	R ²	Waters et al., 1981
3. dominant lethal test	+	I(abs)	Murnik, 1976
4. non-dysjunction and chromosome loss	-	I(abs)	"
mammalian cell cultures:			
1.			
<i>in vivo</i> animal studies:			
1. mouse dominant lethal test	-	R ²	Waters et al., 1981
2. Sister chromatid exchange in CHO cells	-	R	Waters, et al., 1982, cited in CDFA, 1986 ³ .
3. mouse micronucleus test	-	R	" "

in vivo / in vitro Human studies:

1.

plant material assays:

1.

IV. Cell Transformation Assays

Footnotes:

1. Quality assessment is a judgement of the overall validity of the study. A = acceptable (this indicates that useful information is provided), I = inadequate (this indicates flaws in study design, data interpretation, or questionable significance), ? = unable to judge (when uncommon study is used with little basis for comparison or no reference compounds, but apparently acceptable study design and interpretation. May also be used when there is inadequate information to make a judgement), R = signifies that the study was cited in a review, but original study was not evaluated. If the review (e.g. EPA or CDFA) gave a quality rating, this is indicated prior to the R.

2. This reference is a summary of several contract studies performed on Bromacil and other pesticides over several years. The contracts were sponsored by the EPA and performed at SRI International, Menlo Park, CA, and WARF Institute, Inc, Madison WI. This review is apparently written by the EPA contract officer as lead author, with the scientists at SRI and WARF as co-authors. We did not review the original EPA contract reports.

3. California Department of Food and Agriculture (1986). Summary of toxicology Data - Bromacil, SB 950-020, Tolerance #210, dated December 8, 1986.

Review Table for Mutagenicity of Dalapon

<u>TEST ORGANISM TYPE</u>	<u>Response</u>	<u>Quality</u> ¹	<u>Reference</u>
I. Point Mutations or Specific Locus tests			
Microbial assays:			
1. Ames test, strains identified by old nomenclature; -S9	-	I	Anderson et al., 1972
2. Ames TA 98, 100, \pm S9	-	A	Moriya et al., 1983
3. <i>Aspergillus nidulans</i> , 8-azaguanine resist.	-	?	Bignami et al., 1977
4. " " , somatic recombinations	-	?	"
5. " " , non-dysjunction	-	?	"
6. " " , non-dysjunction	-	A	Morpurgo et al., 1979
7. Ames test, TA1535, 1536, 1537, 1538; \pm S9	-	A	Carere et al., 1978
8. <i>S. coelicolor his A1A</i> , forward mutation (S9 ?)	+	?	" "
mammalian cell cultures:			
1.			
<i>in vivo</i> host mediated assays:			
1.			
II. DNA Damage / Repair Tests			
Microbial assays:			
1.			
Mammalian Cell culture:			
1.			
III. Chromosomal Abberations / Cytogenetic tests			
drosophila tests:			
1.			
mammalian cell cultures:			
1. CHO cells \pm S9	-	IR	CDFA, 1986 ²
2. mouse bone marrow, no study details	+	I(abs)	Kurinyi et al., 1982
<i>in vivo</i> animal studies:			
1.			
<i>in vivo</i> / <i>in vitro</i> Human studies:			
1.			
plant material assays:			
1. <i>Pelargonium zonale</i> growth inhibition	+	?	Pohleim et al., 1976
IV. Cell Transformation Assays			

Footnotes:

1. Quality assessment is a judgement of the overall validity of the study. A = acceptable (this indicates that useful information is provided), I = inadequate (this indicates flaws in study design, data interpretation, or questionable significance), ? = unable to judge (when uncommon study is used with little basis for comparison or no reference compounds, but apparently acceptable study design and interpretation. May also be used when there is inadequate information to make a judgement), R = signifies that the study was cited in a review, but original study was not evaluated. If the review (e.g. EPA or CDFA) gave a quality rating, this is indicated prior to the R.

2. California Department of Food and Agriculture, Medical Toxicology Branch, Summary of Toxicology Data - Dalapon SB 950-502, tolerance #150, "super summary" dated May 12, 1986. Most of the above referenced studies were reviewed, but individual rankings were provided for only a few. However, CDFA indicated data gaps, based on "inadequate studies", in all three areas of mutagenicity for dalapon (gene mutation, chromosome and DNA damage).

3. No mutagenicity data were reviewed in EPA Tox one liners.

Summary of Mutagenicity Studies of 2,4-D

<u>TEST ORGANISM TYPE</u>	<u>Response</u>	<u>Quality</u> ¹	<u>Reference</u>
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I. Point Mutations or Specific Locus tests

Microbial assays:

1. Ames TA 98, 100, 1535, 1527, 1538, \pm S9	-	A	Moriya et al, 1983
2. Ames, strains unspecified, - S9	-	I	Anderson et al. 1972
3. " "	-	I	Nagy et al., 1975
4. " "	-	R	Fahrig, 1974
5. Ames TA 98, 100, 1535, 1527, 1538, \pm S9	-	R	Waters et al., 1981b
6. <i>Streptomyces</i>	\pm	A	Zetterberg, 1977
7. <i>Saccharomyces cerevisiae</i>	-	R	Fahrig, 1974
8. <i>Saccharomyces cerevisiae</i> (low pH)	+	A	Siebert & Lemperle, 1974
9. " " "	+	A	Zetterberg et al., 1977
10. " " mitotic recom.	-	A	Waters et al., 1981b

Mammalian cell cultures:

1. Chinese hamster V79 cells	+	A	Ahmed et al., 1977a
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II. DNA Damage / Repair Tests

Microbial assays:

1. <i>E. Coli</i> (WP2 <i>hcr</i>)	-	A	Moriya et al., 1983
2. "	-	I	Ficsor Piccolo, 1972
3. " (WP2 <i>uvr A</i> ⁻)	-	R	Waters et al., 1981b
4. " (Pol A)	+	R	Water et al., 1981b
5. <i>Bacillus subtilis</i>	+	R	Waters et al., 1981b

Mammalian Cell culture:

1. UDS rat hepatocytes	-	A	Probst et al., 1981
2. human fetal lung fibroblasts	-	R	Waters et al., 1981b

Other:

1. inhibition of testicular DNA synthesis	+	R	Seiler, 1979
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III. Chromosomal Abberations / Cytogenetic tests

Drosophila tests:

1. somatic mutations (eye color)	\pm	A	Rasmusson et al., 1976
2. sex-linked recessive lethality	-	?	Vogel & Chandler, 1974
3. " " "	+	A	Magnusson et al., 1977
4. chromosome loss, non-dysj., X-Y recomb.	-	A	Ramel, 1977
5. chromosome loss	-	A	Woodruff et al., 1983

Mammalian cell cultures:

1. human lymphocyte SCE	+	A	Korte & Jalal, 1982
2. CH bone marrow cells	±	A	Linnainmaa, 1984
3. bovine lymphocyte mitogenesis	-	A	McCabe & Nowak, 1986
4. bovine kidney cells & blood cells	±	I	Bongso & Basrur, 1973
5. human embryonic fibroblasts	+	I	Berin et al., cited in Seiler, 1978 (in russian)

In vivo animal studies:

1. rat lymphocyte SCE	-	A	Linnainmaa, 1984
2. CH bone marrow cells	-	A	Linnainmaa, 1984
3. micronucleus/mouse	-	A	Jenssen and Renberg, 1976
4. micronucleus mouse	-	R	Seiler, 1978
5. Dominant lethal test	-	A	Epstein, et. al, 1972
6. chromosomal breakage (mouse)	+	R	three Russian studies, cited in Seiler, 1978

In vivo human studies:

1. Lymphocyte chromosomal abber.	+	I	Yoder et al., 1973
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Plant material assays:

1. <i>Pelargonium zonale</i> mutation	±	?	Pohlheim et al., 1977
2. Chromosomal abber. in <i>Vicia faba</i>	+	?	Amer & Ali, 1974
3. anaphase aberrations in plant cells	-	?	Singh & Harvey, 1975
4. <i>Nicotiana</i> chromosomal aberrations	+	?	Ronchi et al., 1976
5. polyploidy, fragmentation in plants	++	?	Grant, 1973

IV. Cell Transformation

1. SV-40 transformation of human fibroblasts	+	A	Ahmed et al., 1977b
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Footnotes:

1. Quality assessment is a judgement of the overall validity of the study. A = acceptable (this indicates that useful information is provided), I = inadequate (this indicates flaws in study design, data interpretation, or questionable significance), ? = unable to judge (when uncommon study is used with little basis for comparison or no reference compounds, but apparently acceptable study design and interpretation. May also be used when there is inadequate information to make a judgement), R = signifies that the study was cited in a review, but original study was not evaluated. If the review (e.g. EPA or CDFA) gave a quality rating, this is indicated prior to the R.

Review Table for Mutagenicity of 2,4-DP (Dichlorprop)

<u>TEST ORGANISM TYPE</u>	<u>Response</u>	<u>Quality</u> ¹	<u>Reference</u>
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I. Point Mutations or Specific Locus tests

Microbial assays:

1. Ames TA 98, 1537, 1538, \pm S9	-	AR ²	EPA, 1984 ²
2. <i>S. cerevisiae</i> D7, mitotic cross over, no S9	-	AR ²	EPA, 1984 ²
3. <i>S. cerevisiae</i> D7, mitotic gene conversion, no S9	+	AR ²	EPA, 1984 ²
4. <i>S. cerevisiae</i> D7, reverse mutation, no S9	+	AR ²	EPA, 1984 ²

mammalian cell cultures:

1.

in vivo host mediated assays:

1.

II. DNA Damage / Repair Tests

Microbial assays:

1. E coli W3110 & p3478, UDS (?) \pm S9	\pm	AR ²	EPA, 1984 ²
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Mammalian Cell culture:

1.

Other:

1. inhibition of testicular DNA synthesis	-	I	Seiler, 1979
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III. Chromosomal Abberations / Cytogenetic tests

drosophila tests:

1.

mammalian cell cultures:

1.

in vivo animal studies:

1.

in vivo / *in vitro* Human studies:

1.

plant material assays:

1.

IV. Cell Transformation Assays

Footnotes:

1. Quality assessment is a judgement of the overall validity of the study. A = acceptable (this indicates that useful information is provided), I = inadequate (this indicates flaws in study design, data interpretation, or questionable significance), ? = unable to judge (when uncommon study is used with little basis for comparison or no reference compounds, but apparently acceptable study design and interpretation. May also be used when there is inadequate information to make a judgement), R = signifies that the study was cited in a review, but original study was not evaluated. If the review (e.g. EPA or CDFA) gave a quality rating, this is indicated prior to the R.

2. EPA Tox One-liner, Tox Chem No. 320 - 2,4-DP, dated 8/20/84.

Review Table for Mutagenicity of Dicamba

TEST ORGANISM TYPEResponseQuality¹Reference

I. Point Mutations or Specific Locus tests

Microbial assays:

- | | | | |
|---|---|------------------|------------------------|
| 1. Ames TA 98, 100, 1535, 1537, 1538, \pm S9 | - | A | Eisenbeis et al., 1981 |
| 2. Ames TA 98, 100, \pm S9 | - | A | Moriya et al., 1983 |
| 3. Ames Test, strains identified by old nomenclature; -S9 | - | I | Andersen et al., 1972 |
| 4. rII mutants of T ₄ Bacteriophage | - | I | " " " |
| 5. Ames test, TA 100, 1535, 1537, 1538, +mouse S9 | - | AR ³ | Waters et al., 1981 |
| 6. <i>S. cerevisiae</i> D3 recombination | - | I,R ³ | " |

mammalian cell cultures:

1.

in vivo host mediated assays:

1.

II. DNA Damage / Repair Tests

Microbial assays:

- | | | | |
|---|---|------------------|---------------------|
| 1. <i>B. subtilis</i> H17 and M45 rec+ | + | A,R ³ | Waters et al., 1981 |
| 2. <i>E. Coli</i> WP2 <i>uvrA</i> ⁻ | - | A,R ³ | " |
| 3. <i>E. Coli</i> W3110 & P3478 repair deficient, (Pol A) | + | A,R ³ | " |

Mammalian Cell culture:

- | | | | |
|---|---|-------------------|--------------------------|
| 1. Unscheduled DNA synthesis, primary rat hepatocytes | - | AR ^{4,5} | EPA, 1986;
CDFA, 1986 |
|---|---|-------------------|--------------------------|

III. Chromosomal Abberations / Cytogenetic tests

drosophila tests:

- | | | | |
|-------------------------------------|-------|------------------|---------------------|
| 1. sex-linked recessive lethal test | \pm | A,R ³ | Waters et al., 1981 |
|-------------------------------------|-------|------------------|---------------------|

mammalian cell cultures:

1.

in vivo animal studies:

1.

in vivo / *in vitro* Human studies:

1.

plant material assays:

- | | | | |
|------------------------------------|---|---|-----------------|
| 1. tradescantia-micronucleus tests | + | ? | Ma et al., 1984 |
|------------------------------------|---|---|-----------------|

IV. Cell Transformation Assays

Footnotes:

1. Quality assessment is a judgement of the overall validity of the study. A = acceptable (this indicates that useful information is provided), I = inadequate (this indicates flaws in study design, data interpretation, or questionable significance), ? = unable to judge (when uncommon study is used with little basis for comparison or no reference compounds, but apparently acceptable study design and interpretation. May also be used when there is inadequate information to make a judgement), R = signifies that the study was cited in a review, but original study was not evaluated. If the review (e.g. EPA or CDFA) gave a quality rating, this is indicated prior to the R.

2. This extensive study of herbicide mutagenicity was conducted by SRI International and WARF Research Institute under contract to EPA. The results of each of these studies have been reported in numerous forms, including the book citation listed, as well as in the *Journal of Environmental Science and Health*, B15(6), 867-906, 1980. These same studies have also been evaluated by CDFA and EPA (see notes 3 and 4 below), and have been judged to be adequate, with the exception of the yeast mutation assay (D3 recombination) in which no positive control was reported.

3. CDFA, California Department of Food and Agriculture, Medical Toxicology Branch, Summary of Toxicology Data - Dicamba, SB950 - 070, tolerance #227, dated August 8, 1986.

4. EPA Tox one liners, No. 295 - Dicamba, dated 06/11/85.

Review Table for Mutagenicity of Diuron

<u>TEST ORGANISM TYPE</u>	<u>Response</u>	<u>Quality</u> ¹	<u>Reference</u>
I. Point Mutations or Specific Locus tests			
Microbial assays:			
1. Ames TA 98, 100, 1535, 1537, 1538, \pm S9	-	A	Moriya et al, 1983
2. Ames, strains identified by old nomenclature; - S9	-	I	Anderson et al., 1972
3. Ames test, strains unidentified, - S9	-	R	Fahrig, 1973
4. Ames test, strains unspecified, \pm S9	-	IR ² AR ³	EPA, 1986; CDFA, 1986
mammalian cell cultures:			
1. CHO/HGPRT forward mutation assay	-	AR ^{2,3}	EPA, 1986; CDFA, 1986
<i>in vivo</i> host mediated assays:			
1.			
II. DNA Damage / Repair Tests			
Microbial assays:			
1.			
Mammalian Cell culture:			
1. Unscheduled DNA synthesis, primary rat hepatocytes	-	AR ² IR ^{3,4}	EPA, 1986; CDFA, 1986
III. Chromosomal Abberations / Cytogenetic tests			
drosophila tests:			
1.			
mammalian cell cultures:			
1.			
<i>in vivo</i> animal studies:			
1. rat bone marrow cells	\pm	AR ^{2,3}	EPA, 1986 CDFA, 1986
<i>in vivo</i> / <i>in vitro</i> Human studies:			
1.			
plant material assays:			
1.			

IV. Cell Transformation Assays

Footnotes:

1. Quality assessment is a judgement of the overall validity of the study. A = acceptable (this indicates that useful information is provided), I = inadequate (this indicates flaws in study design, data interpretation, or questionable significance), ? = unable to judge (when uncommon study is used with little basis for comparison or no reference compounds, but apparently acceptable study design and interpretation. May also be used when there is inadequate information to make a judgement), R = signifies that the study was cited in a review, but original study was not evaluated. If the review (e.g. EPA or CDFA) gave a quality rating, this is indicated prior to the R.

2. EPA Tox one liners, No. TL-9, Diuron, dated 10/14/86

3. CDFA (California Department of Food and Agriculture), Medical Toxicology Branch, Summary of Toxicology Data - Diuron, SB 950-018, Tolerance # 106, dated December 8, 1986.

4. CDFA classified this study as inadequate because pages were missing from the report at the time of review, so no review was completed.

Review Table for Mutagenicity of Fosamine

<u>TEST ORGANISM TYPE</u>	<u>Response</u>	<u>Quality</u> ¹	<u>Reference</u>
---------------------------	-----------------	-----------------------------	------------------

I. Point Mutations or Specific Locus tests

Microbial assays:

- | | | | |
|--|---|----|-------------------------|
| 1. Ames TA 98, 100, 1535, 1537, 1538, \pm S9 | - | IR | CDFA, 1986 ² |
| 2. Ames TA 98, 100, 1535, 1537, 1538, \pm S9 | - | A | Moriya et al., 1983 |

mammalian cell cultures:

- | | | | |
|--------------|---|----|---|
| 1. CHO/HGPRT | - | AR | CDFA, 1986 ² ,
EPA, 1986 ³ |
|--------------|---|----|---|

in vivo host mediated assays:

- 1.

II. DNA Damage / Repair Tests

Microbial assays:

- | | | | |
|----------------------------------|---|---|---------------------|
| 1. <i>E. coli</i> WP2 <i>hcr</i> | - | A | Moriya et al., 1983 |
|----------------------------------|---|---|---------------------|

Mammalian Cell culture:

- | | | | |
|------------------------|---|---------------------------------|---|
| 1. rat hepatocytes UDS | - | I ² A ³ R | CDFA, 1986 ² ,
EPA, 1985 ³ |
|------------------------|---|---------------------------------|---|

III. Chromosomal Abberations / Cytogenetic tests

drosophila tests:

- 1.

mammalian cell cultures:

- | | | | |
|------------------------------------|---|---------------------------------|---|
| 1. Chinese hamster cells, \pm S9 | + | I ² A ³ R | CDFA, 1986 ² ,
EPA, 1985 ³ |
|------------------------------------|---|---------------------------------|---|

in vivo animal studies:

- | | | | |
|---|---|---------------------------------|---|
| 1. rat cytogenetics, tissue unspecified | - | I ² A ³ R | CDFA, 1986 ² ,
EPA, 1985 ³ |
|---|---|---------------------------------|---|

in vivo / *in vitro* Human studies:

- 1.

plant material assays:

- 1.

IV. Cell Transformation Assays

Footnotes:

1. Quality assessment is a judgement of the overall validity of the study. A = acceptable (this indicates that useful information is provided), I = inadequate (this indicates flaws in study design, data interpretation, or questionable significance), ? = unable to judge (when uncommon study is used with little basis for comparison or no reference compounds, but apparently acceptable study design and interpretation. May also be used when there is inadequate information to make a judgement), R = signifies that the study was cited in a review, but original study was not evaluated. If the review (e.g. EPA or CDFA) gave a quality rating, this is indicated prior to the R.

2. California Department of Food and Agriculture, Medical Toxicology Branch, Summary of Toxicology Data - Fosamine, SB 950-312, Tolerance # 50097, dated August 6, 1986

3. EPA Tox one liners, No. 465G-Fosamine, dated 7/14/85.

4. An exhaustive computer-based search of the literature revealed only a single published study of fosamine mutagenicity.

Review Table for Mutagenicity of Glyphosate

<u>TEST ORGANISM TYPE</u>	<u>Response</u>	<u>Quality</u> ¹	<u>Reference</u>
I. Point Mutations or Specific Locus tests			
Microbial assays:			
1. Ames assay, TA 100, 98, 1535, 1537, 1538 \pm S9	-	A	Moriya et al., 1983
2. Ames test, TA100, 98, 1535, 1537, 1538 +S9	-	A	Long & Li, 1987 ²
drosophila tests:			
1.			
mammalian cell cultures			
1. CHO/HGPRT forward mutation, \pm S9	-	A	Long & Li, 1987
<i>in vivo</i> host mediated assays:			
1. host mediated, rats and mice	-	I, R	EPA, 1986 ³
II. DNA Damage / Repair Tests			
Microbial assays			
1. <i>B. Subtilis</i> H17 (rec+), M45 (rec-); recombination	-	A, (I ⁴)	Long & Li, 1987 ²
2. <i>E. coli</i> WP2 <i>hcr</i>	-	A	Moriya et al., 1983
Mammalian Cell culture:			
1. UDS in hepatocytes	\pm^5	A(I ⁵)	Long & Li, 1987
III. Chromosomal Abberations / Cytogenetic tests			
mammalian cell cultures:			
1.			
<i>in vivo</i> animal studies:			
1. mouse bone marrow, chromosome abberations	-	A	Long & Li, 1987
2. mouse dominant lethal	-	A(I ⁶)	Long & Li, 1987
<i>in vivo / in vitro</i> Human studies:			
1. Sister chromatid exchange in cultured lymphocytes	+	I	Vigfusson & Vyse 1980
2.			
plant material assays:			
1.			
IV. Cell Transformation Assays			

Footnotes:

1. Quality assessment is a judgement of the overall validity of the study. A = acceptable (this indicates that useful information is provided), I = inadequate (this indicates flaws in study design, data interpretation, or questionable significance), ? = unable to judge (when uncommon study is used with little basis for comparison or no reference compounds, but apparently acceptable study design and interpretation. May also be used when there is inadequate information to make a judgement), R = signifies that the study was cited in a review, but original study was not evaluated. If the review (e.g. EPA or CDFA) gave a quality rating, this is indicated prior to the R.
2. This reference is cited as an abstract presented at the 1987 meeting of the Society of Toxicology. However, descriptions of methods and actual results were provided by the author for our independent review. A manuscript of this work is currently in preparation and will be submitted for publication in a peer-reviewed journal.
3. Environmental Protection Agency, (1986). EPA-Tox one liners, No. 661A - Glyphosate, dated 04/08/86.
4. CDFA, 1986: California Department of Food and Agriculture, Chemical Toxicology Branch, Summary Review of Glyphosate, dated Dec. 2, 1986. This particular study was judged "in complete, unacceptable" because "only single plates per treatment, doses tested from 20 - 200 µg/disk but reported as 20 - 2000". However, data provided to us shows full range to to 2000 µg per plate. EPA³ has accepted this study as "core minimal".
5. CDFA judged this study to be "because authors did not provide justification for "dismissing the high dose result ... without further information". Although the authors reported no effect, the highest dose did show a small but significant increase in nuclear grains. Thus, we report the effects as ±, as there was no indication of any effect except at the highest dose (0.125 mg/ml). the design and the conduct of the assay appear adequate. The EPA³ has classified this study as negative study with "core acceptable" grade.
6. CDFA judges this study to be "Incomplete, unacceptable" because "too few animals, individual data missing". We believe 10 males per treatment group, mated to 2 females for 8 weeks post-dosing is an adequate number of animals to be of some scientific value. The data we were provided did not indicate that any data were missing, although original records were not provided. Never-the-less, the information provided us indicates that all animals are accounted for, and appears to be adequately conducted and reported, and thus is judged to be scientifically valid. EPA³ has accepted this negative study as a "core minimum" .

Review Table for Mutagenicity of Hexazinone

<u>TEST ORGANISM TYPE</u>	<u>Response</u>	<u>Quality</u> ¹	<u>Reference</u>
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I. Point Mutations or Specific Locus tests

Microbial assays:

1. Ames TA 98, 100, 1535, 1537, 1538, \pm S9	-	AR	EPA, 1984 ²
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mammalian cell cultures:

1.

in vivo host mediated assays:

1.

II. DNA Damage / Repair Tests

Microbial assays:

1.

Mammalian Cell culture:

1. rat hepatocytes UDS	-	AR	EPA, 1984 ²
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III. Chromosomal Abberations / Cytogenetic tests

drosophila tests:

1.

mammalian cell cultures:

1. Chinese hamster cells, without S9	+	AR	EPA, 1984 ²
2. Chinese hamster cells, with S9	+	AR	EPA, 1984 ²

in vivo animal studies:

1. rat bone marrow cytogenetic	-	AR	EPA, 1984 ²
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in vivo / *in vitro* Human studies:

1.

plant material assays:

1.

IV. Cell Transformation Assays

Footnotes:

1. Quality assessment is a judgement of the overall validity of the study. A = acceptable (this indicates that useful information is provided), I = inadequate (this indicates flaws in study design, data interpretation, or questionable significance), ? = unable to judge (when uncommon study is used with little basis for comparison or no reference compounds, but apparently acceptable study design and interpretation. May also be used when there is inadequate information to make a judgement), R = signifies that the study was cited in a review, but original study was not evaluated. If the review (e.g. EPA or CDFA) gave a quality rating, this is indicated prior to the R.

2. EPA Tox one liners, No. 271 AA - Hexazinone, dated 9/20/84. Hexazinone Registration Standard, dated February, 1982, indicates that no further mutagenicity testing is required for hexazinone under FIFRA (Table A, p. 3-9).

3. An exhaustive computer based search of the literature revealed no published studies of hexazinone mutagenicity. No California Department of Food and Agriculture review was available for Hexazinone.

Review Table for Mutagenicity of Picloram (Tordon)

<u>TEST ORGANISM TYPE</u>	<u>Response</u>	<u>Quality</u> ¹	<u>Reference</u>
I. Point Mutations or Specific Locus tests			
Microbial assays:			
1. Ames test, TA1535, 1536, 1537, 1538; ± S9 (EPA ³ has classified this study as "partially satisfying gene mutation requirement")	-	A (I ²)	Carere <i>et al.</i> , 1978
2. <i>S. coelicolor his</i> A1A, forward mutation (S9 ?)	+	? (I ²)	Carere <i>et al.</i> , 1978
3. <i>Aspergillus nidulans</i> , non-dysjunction	-	? (I ²)	Bignami <i>et al.</i> , 1977
4. <i>Aspergillus nidulans</i> , non-dysjunction	-	A (I ²)	Morpurgo <i>et al.</i> , 1979
5. <i>S. cerevisae</i> (unspecified)	+	I	L'Vova, 1984
6. <i>S. cerevisae</i> (unspecified)	+	I	Guerzoni <i>et al.</i> , 1976, cited in Dow Technical Data Sheet ⁴
7. Ames test, 8 strains unspecified, - S9	-	I	Anderson <i>et al.</i> , 1972
8. Ames test, TA100, 98, 1535, 1537; +S9 (this study is part of NTP-sponsored study with good quality assurance protocols)	-	A	Mortelmans <i>et al.</i> , 1986

mammalian cell cultures:

1. mouse bone marrow cells (unspecified test)	-	I	L'Vova, 1984
---	---	---	--------------

in vivo host mediated assays:

1.

II. DNA Damage / Repair Tests

Microbial assays:

1.

Mammalian cell culture:

1.

III. Chromosomal Abberations / Cytogenetic tests

drosophila tests:

1. complete and partial chromosome loss, <i>mus</i> -302 repair defective females	-	A	Woodruff <i>et al.</i> , 1983
--	---	---	-------------------------------

mammalian cell cultures:

1. human peripheral lymphocytes (unspecified test)	-	I	L'Vova, 1984
--	---	---	--------------

in vivo animal studies:

1. rat bone marrow cells	-	I,R	Johnston <i>et al.</i> , 1976 (also CDFA ³ , 1986)
--------------------------	---	-----	--

in vivo Human studies:

1. lymphocyte chromosome aberrations in applicators + I Yoder et al., 1973.

plant material assays:

- 1.

IV. Cell Transformation Assays

mammalian cell cultures:

- 1.

Footnotes:

1. Quality assessment is a judgement of the overall validity of the study. A = acceptable (this indicates that useful information is provided), I = inadequate (this indicates flaws in study design, data interpretation, or questionable significance), ? = unable to judge (e.g. when an unusual test system is used with little basis for comparison or no reference compounds, but apparently acceptable in design and interpretation. May also be used when there is inadequate information to make a judgement), R = signifies that the study was cited in a review, and original study was not evaluated. If a review (e.g. EPA or CDFA) gave a quality rating, this is indicated prior to the R. In certain circumstances, there may be disagreement between our evaluations and those of others. If this occurs, two contradictory ratings may appear, with the rating and source of the contradictory evaluation in parentheses.

2. California Department of Food and Agriculture, Toxicology Branch. Summary of Data - Picloram, SB950-306, Tolerance #292, July 30, 1986. Details of why studies were judged inadequate by CDFA were not provided, so no rational comparison of CDFA judgements with ours can be made.

3. EPA, 1985. Guidance for the Reregistration of pesticide products containing Picloram as the active ingredient. EPA case number 0096, March, 1985.

4. Dow Chemical Company Technical Data Sheet, undated, #137-1640-1183.

5. No "EPA-one liners" were available to compare EPA's "core mutagenicity" evaluations of these studies with ours or CDFA's.

Review Table for Mutagenicity of Simazine

<u>TEST ORGANISM TYPE</u>	<u>Response</u>	<u>Quality</u> ¹	<u>Reference</u>
I. Point Mutations or Specific Locus tests			
Microbial assays:			
1. Ames TA98, 199, 1535, 1537, \pm S9	-	I R	CDFA, 1986 ²
2. <i>S. cerevisiae</i> D3 and D7 assays, \pm S9	-	I(abs)	Riccio et al., 1981
3. Ames test, TA 100, 1535, 1537, 1538, +mouse S9	-	R ³	Waters et al., 1981
4. <i>S. cerevisiae</i> D3 recombination	-	R ³	"
mammalian cell cultures:			
1. mouse lymphoma L5178Y TK+/-, \pm S9	-	I R	CDFA, 1986 ²
2. UDS in human fetal lung fibroblasts (WI-38 cells)	-	R ³	Waters et al., 1981
<i>in vivo</i> host mediated assays:			
1. <i>Salmonella</i> host mediated assay in mice	-	?R	CDFA, 1986 ²
II. DNA Damage / Repair Tests			
Microbial assays:			
1. <i>B. subtilis</i> H17 and M45 rec+	-	R ³	Waters et al., 1981
2. <i>E. Coli</i> WP2 <i>uvrA</i> ⁻	-	R ³	"
3. <i>E. Coli</i> W3110 & P3478 repair deficient, (Pol A)	-	R ³	"
Mammalian Cell culture:			
1. primary rat hepatocytes UDS	-	AR	CDFA, 1986 ²
2. DNA repair (UDS), human fibroblasts, no S9	-	I R	CDFA, 1986 ²
III. Chromosomal Abberations / Cytogenetic tests			
drosophila tests:			
1. sex-linked recessive lethal	\pm	?	Valencia, R. 1981
2. sex-linked recessive lethal	+	R ³	Waters et al., 1981
3. dominant lethal mutations	+	I(abs)	Murnick, 1976
mammalian cell cultures:			
1. Chinese hamsters, micronucleus test	-	I R	CDFA, 1986 ²
<i>in vivo</i> animal studies:			
1.			
<i>in vivo</i> / <i>in vitro</i> Human studies:			
1. cultured human lymphocytes, SCE	+	I	Ghiazza et al., 1984
plant material assays:			
1. <i>Pelargonium zonale</i> chlorophyll defects	+	I(abs)	Pohleim et al., 1976

2. *Vicia* root tip SCE and abberations + R Ma, 1982

IV. Cell Transformation Assays

Footnotes:

1. Quality asessment is a judgement of the overall validity of the study. A = acceptable (this indicates that useful information is provided), I = inadequate (this indicates flaws in study design, data interpretation, or questionable significance), ? = unable to judge (when uncommon study is used with little basis for comparison or no reference compounds, but apparently acceptable study design and interpretation. May also be used when there is inadequate information to make a judgement), R = signifies that the study was cited in a review, but original study was not evaluated. If the review (e.g. EPA or CDFA) gave a quality rating, this is indicated prior to the R.

2. California Department of Food and Agricuture, Medical Toxicology Branch, Summary of Toxicology Data - Simazine SB950-129, Tolerance #213, dated August 11, 1986.

3. This reference is a summary of several contract studies performed on Simazine and other pesticides over several years. The contracts were sponsored by the EPA and performed at SRI International, Menlo Park ,CA, and WARF Institute, Inc, Madison WI. This review is apparently written by the EPA contract officer as lead author, with the scientists at SRI and WARF as co-authors. We did not review the original EPA contract reports.

Review Table for Mutagenicity of Tebuthiuron

<u>TEST ORGANISM TYPE</u>	<u>Response</u>	<u>Quality</u> ¹	<u>Reference</u>
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I. Point Mutations or Specific Locus tests

Microbial assays:

1. Ames TA 98, 100, 1535, 1537, 1538, \pm S9	-	AR	EPA, 1986 ²
--	---	----	------------------------

mammalian cell cultures:

1. mouse lymphoma cell forward mutation	\pm	AR	EPA, 1986 ²
---	-------	----	------------------------

in vivo host mediated assays:

1.

II. DNA Damage / Repair Tests

Microbial assays:

1.

Mammalian Cell culture:

1. primary rat hepatocytes	?	IR	EPA, 1986 ²
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III. Chromosomal Abberations / Cytogenetic tests

drosophila tests:

1.

mammalian cell cultures:

1.

in vivo animal studies:

1. Chinese hamster bone marrow SCE	?	IR	EPA, 1986 ²
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in vivo / *in vitro* Human studies:

1.

plant material assays:

1.

IV. Cell Transformation Assays

Footnotes:

1. Quality assessment is a judgement of the overall validity of the study. A = acceptable (this indicates that useful information is provided), I = inadequate (this indicates flaws in study design, data interpretation, or questionable significance), ? = unable to judge (when uncommon study is used with little basis for comparison or no reference compounds, but apparently acceptable study design and interpretation. May also be used when there is inadequate information to make a judgement), R = signifies that the study was cited in a review, but original study was not evaluated. If the review (e.g. EPA or CDFA) gave a quality rating, this is indicated prior to the R.

2. EPA-Tox one-liner, number 366AA - tebuthiuron, dated 10/31/86. this is the only source of information on mutagenicity of tebuthiuron available to us for this review.

3. An exhaustive computer-based literature search did not reveal any published mutagenicity studies on tebuthiuron. No California Department of Agriculture review is available.

Review Table for Mutagenicity of Triclopyr (Garlon)

<u>TEST ORGANISM TYPE</u>	<u>Response</u>	<u>Quality¹</u>	<u>Reference</u>
I. Point Mutations or Specific Locus tests			
Microbial assays:			
1. Ames test, TA 98, 100, 1537, 1538, \pm S9	-	AR	EPA, 1985 ² ; CDFA, 1986 ³
2. Ames test, TA 98, 100 (different laboratory)	-	I ³ A ² R	EPA, 1985 ² ; CDFA, 1986 ³
3. Ames test, TA 98, 100, \pm S9	-	A	Moriya et al., 1983
mammalian cell cultures:			
1.			
<i>in vivo</i> host mediated assays:			
1. male mice, <i>Salmonella</i> TA1530 and G46,	?	I	CDFA, 1986 ³
2. male mice, <i>Saccharmyces</i> D3	?	I	CDFA, 1986 ³
II. DNA Damage / Repair Tests			
Microbial assays:			
1. <i>B. subtilis</i> H17/M45 recombination	-	I ³ A ² R	EPA, 1985 ² ; CDFA, 1986
Mammalian Cell culture:			
1. primary rat hepatocytes	-	A	CDFA, 1986 ³
III. Chromosomal Abberations / Cytogenetic tests			
drosophila tests:			
1. rat cytogenetic	-	I ³ A ² R	EPA, 1985 ² ; CDFA, 1986 ³
mammalian cell cultures:			
1.			
<i>in vivo</i> animal studies:			
1. mouse, dominant lethal assay	-	A, R	EPA, 1985 ² ; CDFA, 1986 ³
2. rat, dominant lethal	\pm	I ³ A ² R	EPA, 1985 ² ; CDFA, 1986 ³
3. mouse micronucleus test	-	A	CDFA, 1986 ³
<i>in vivo</i> / <i>in vitro</i> Human studies:			
1.			

plant material assays:

1.

IV. Cell Transformation Assays

Footnotes:

1. Quality assessment is a judgement of the overall validity of the study. A = acceptable (this indicates that useful information is provided), I = inadequate (this indicates flaws in study design, data interpretation, or questionable significance), ? = unable to judge (when uncommon study is used with little basis for comparison or no reference compounds, but apparently acceptable study design and interpretation. May also be used when there is inadequate information to make a judgement), R = signifies that the study was cited in a review, but original study was not evaluated. If the review (e.g. EPA or CDFA) gave a quality rating, this is indicated prior to the R.

2. EPA Tox-one liner, No. 882-I, Garlon, Dated 2/7/85. Evaluations for mutagenicity were all ranked acceptable, supplementary or minimum.

3. California Department of Food and Agriculture, Medical Toxicology Branch, Summary of Toxicology data for Triclopyr, SB 950-227, tolerance #417, dated October 27, 1986. All of the above studies were thoroughly reviewed and ranked as indicated above. In their summary for mutagenicity, CDFA showed "no data gap" for all mutagenicity categories.

4. An exhaustive computer-based literature search revealed only one published mutagenicity study with triclopyr.

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SUMMARY OF ONCOGENICITY STUDIES OF HERBICIDES¹

<u>Species</u>	<u>Dose</u>	<u>Route</u>	<u>Time</u>	<u>#/group</u>	<u>Effect</u> ²	<u>Quality</u> ³	<u>Study</u>
<u>Amitrole</u>							
Rat	0, 10, 50, 100, 500 ppm	diet	2 y	?	thyroid adenomas at 50, 100, 500 ppm	I	Hazelton, '59
Rat	0, 1, 10, 100 ppm	diet	2 y	75	thyroid adenomas in 100 ppm; pituitary adenomas and carcinomas at 100 ppm	M	Steinhoff, '83
mice	0, 1, 10, 100 ppm	diet	2 y	?	increase thyroid weight at 100 ppm; no tumors	M	"
mice	1000 mg/kg/day; 2192 ppm	gav. diet	day 7-28 18 m	18	liver tumors; thyroid tumors (shortened lifespan)	I	Innes, '69
Hams.	0, 1, 10, 100 ppm	diet	2 year		no effects reported	M	Steinhoff, '83
Rat	20-25 mg/kg/day	water	10 - 32 m	55(?)	increased thyroid tumors, increased liver tumors	I	Napulkov, '69
Rat	250 - 5000 ppm	diet	10 - 32 m	52(?)	increased thyroid tumors, increased liver tumors	I	"
Rat	1, 3, 5, 10 ppm; 20, 60, 100, 100 ppm, respectively	diet diet	39 w then 78 w	75	increased thyroid tumors; increased pituitary tumors in high (10 + 100 ppm) group only	M	FDA, '81
Rat	?	inhal	?	?	increased thyroid tumors	I	Johnson, '81
<u>Asulam</u>							
mice	0, 1500, 5000 ppm	diet	18 m	60	questionable incidence of skin and subcutaneous undifferentiated sarcomas at 5000 ppm (rated as "guideline" by EPA)	A	Rhodia, Inc, '78
rats	0,1000, 5000 25000 ppm	diet	107 w	65	follicular thyroid hyperplasia at 5000 and 25000 ppm; possible pheochromocytoma in 25000 ppm males.	M	Huntingdon, '81

<u>Species</u>	<u>Dose</u>	<u>Route</u>	<u>Time</u>	<u>#/group</u>	<u>Effect</u>	<u>Quality</u>	<u>Study</u>
<u>Atrazine</u>							
Rat	0, 1, 10, 100, 1000 ppm	diet	2 yr	?	no apparent effect; poor survival from infections	I	see Crump, '86
Rat	0, 10, 100, 1000 ppm	diet	2 yr	?	no apparent effect; histopathology incomplete	I	see Crump, '86
mice	21.5 m/k/d 82 ppm	gav diet	3 w 18 m	18	no apparent effect; insufficient detail to evaluate	I	Innes, '69
Rat	0, 10, 70, 500, 1000 ppm	diet	2 y	70	dose related increase in female mammary adenocarcinomas (min. 70 ppm) and fibroadenomas (500 ppm); testicular interstitial tumors increased at 1000 ppm; non-oncogenic NOEL set at 70 ppm.	A	Toxicogenics, '86
<u>Bromacil</u>							
rats	0, 50, 250, 1250 ppm	diet	2 y	36	possible adverse effect on thyroid; systemic NOEL 250 ppm; no tumors reported; CDFA states "inadequate evaluation."	M	Haskell, '66
mice	0, 250, 1250, 5000 ppm	diet	18 m	80	increased liver adenomas + carcinomas and liver damage at 5000 ppm; testicular atrophy at all doses - systemic NOEL < 250 ppm; doses in excess of MTD.	M	Haskell, '80
<u>Dalapon</u>							
Rats	0, 100, 300, 1000 ppm	diet	2 y	24 m/20 f	no adverse effect apparent (Data published in summary form in 1960)	M	Hazelton, '56 (Paynter, 1960)
mice	0, 2, 60, 200 mg/kg/day	diet	2 y	50	no oncogenic effects reported; NOEL set at 60 mg/kg/day	A(M)	Dow, '83

Species Dose Route Time #/group Effect Quality Study

2,4-D

rat	0, 1, 5, 15, 45 mg/kg/day	diet	2 year	60	chronic NOEL set at 1 mg/kg/day (kidney effect at 5 mg/kg/day); increased glial cell brain tumors in high dose males; of questionable statistical significance (CDFA - "distribution with clustering in high dose group is most likely by chance based on a number of criteria for significance")	A	Hazelton, '86
mice	47, 100 m/k/d 149, 323 ppm (used acid, isopropyl, isobutyl or n-octyl esters)	gav diet	d 7- 28 74 w	?	no significant increase in tumors	M	Innes , '69
mice	same study as above - reinterpreted				increase in some tumors (reticulum cell sarcoma, neoplasms of liver and lung) with ester formulations, but not 2,4-D acid.	M	Reuber, '83
rats	0, 5, 25, 125, 625, 1250 ppm	diet	2 y	25	no specific target organ tumors; dose related increase in total and malignant tumors, but only 1250 ppm dose was significant (6, 8, 7, 8, 14 tumors in control and consecutive doses, respectively)	M	Hansen, '71
rats	same study as above - reinterpreted*				increase in total malignant tumors, lympho-sarcomas and mammary tumors in females	M	Reuber, '83
rats	1/10 LD50	diet	27 m	120 m/ 45 f	no significant carcinogenic effects with 2,4-D amine	I	Archipov & Kozlova, '74
mice	1/10 LD50	diet	27 m	100 f	no significant carcinogenic effects reported	I	"
mice	skin painting with 2,4-D amine following 3-MC				significant increase in skin papillomas	I	"

* using the same assumptions and model, Crump et al (1986) found that Reuber's interpretation of data resulted in a 3 fold increase in risk compared with NCI interpretation.

<u>Species</u>	<u>Dose</u>	<u>Route</u>	<u>Time</u>	<u>#/group</u>	<u>Effect</u>	<u>Quality</u>	<u>Study</u>
<u>Dicamba</u>							
rats	0, 5, 50, 100, 250, 500 ppm	diet	2 y	32	multiple tissue lesions at all doses, but not dose related; increase in malignant neoplasms at 5, 250 & 500, but not dose-related.	I	Kettering, '62
rats	0, 50, 250, 2500 ppm	diet	2+ y	60	increase in thyroid parafollicular cell carcinoma & malignant lymphoma, high dose males only	A	IRDC, '85
mice	0, 100, 1000, 10000 ppm	diet	2 y	60	possible increase in angiosarcoma at 1000 ppm males; IBT test; not yet validated by EPA	I	IBT, '80
<u>Diuron</u>							
rats	0, 25, 125, 250, 2500 ppm	diet	2 y	35	effects on bone marrow erythropoiesis at high dose; no oncogenic effects reported, but histopathology was stated by CDFA to be inadequate/incomplete	M	U. Rochester, '64
<u>2,4-DP</u>							
rats	0, 25, 50, 250/150 m/k/d (switched at 60 weeks)	diet	2 y	?	dose-related increase in pituitary and thyroid medullary tumors at all doses (in males) ; oncogenic NOEL < 25 m/k/d - increase in rare malignant brain tumors at low dose only (classified as "guideline" by EPA; EPA one-liner only available information; CDFA did not review).	A	CDC Res., '80
mice	0, 25, 100, 300 m/k/d	diet	18 mo	?	no oncogenic response noted; oncogenic NOEL >300 m/k/d; systemic NOEL (liver effects) 100 m/k/d. (this study classified as "guideline" by EPA; EPA one-liner is only available information on this study)	A	CDC Res., '80
<u>Fosamine</u>							
no studies available							

Glyphosate

rat	0, 3, 10, 32 mg/kg/day	diet	2+ y	50	possible increase in testicular tumors in high dose group, but questionable significance; NOEL > 32 mg/kg/day, MTD never reached; (rated "supplemental" by EPA)	M	BioDynamics, '81
mice	0, 1000, 5000, 30,000 ppm	diet	2 y	50	possible increase in renal adenomas in high dose males compared with concurrent control initially reported; re-evaluation of histopathology resulted in no signif. increase in renal tumors.	A	BioDynamics, '81

Hexazinone

rat	0, 200, 1000, 2500 ppm	diet	2 y	36	no apparent oncogenic effects; NOEL (decreased body wt) set at 200 ppm. (satisfies EPA requirem.)	A	Haskell Lab., '77
mice	0, 200, 2500, 10000 ppm	diet	2 y	80	effects on liver at 2500 and 10000 ppm; no oncogenic effects reported.	A	IRDC, '81

Picloram

rats	0, 7437, 14875 ppm	diet	80 w	50	no treatment related increases in any tumors in males; increase in benign liver tumors in high dose females	M	NCI, '78
rats	re-review of above study*				high incidence of malignant tumors (adrenal, pituitary, liver, mammary and thyroid); tumors in control animals were unusually high	M	Reuber, '81
mice	0, 2531, 5062 ppm	diet	80 w	50	no apparent oncogenic effects reported	M	NCI, '78
mice	re-review of above study*				increased neoplasms of spleen in high dose group	M	Reuber, '81
rats	0, 20, 60, 200 mg/kg/day	diet	2 y	70	mild hepatic hypertrophy at 60 and 200 mg/kg/d; no oncogenic effects reported. This study judged adequate by both EPA and CDFA.	A	Dow, '86

* using the same assumptions and model, Crump et al (1986) found that Reuber's interpretation of data resulted in a 6 fold increase in risk compared with NCI interpretation.

Simazine

rats 0, 1, 10, 100 ppm diet 2 y 30 no observed effects; NOEL set at > 100 ppm I Hazelton '60

mice 2 ppm solution of ip inj increased incidence of malignant lymphoma I Donna, '81
25% atrazine:
37.5% simazine

Tebuthiuron

rats 0, 400, 800, 1600 ppm diet 2 y 40 vacuolization of pancreatic acinar cells at high dose; no oncogenic effect reported; systemic NOEL set at 400 ppm (20 mg/kg/day) M Lilly Res, '76

mice 0, 400, 800 1600 ppm diet 2 y 40 no evidence of toxicity or oncogenicity M Lily Res., '76

Triclopyr (Garlon)

rats 0, 3, 10, 30 m/k/d diet 2 y 50 no apparent oncogenic effects M IBT, '78

mice 0, 24, 80, 240 ppm diet 2y 50 questionable increase in benign lung tumors at 24 & 240 ppm in males; in females at 240 ppm; significance depends upon control group used. Independent evaluation concluded that lung tumor effect "could not be substantiated." M Dow, '79

- 1 Data reviewed for this table were obtained primarily from notes of the California Department of Food and Agriculture (CDFA) review of these herbicides. CDFA notes were available for all herbicides except Amitrole. In addition to CDFA documents, EPA reregistration standards and other documents that serve as the source for EPA "one-liners" were used, as well as evaluations in the Crump et al., 1986 review of herbicides for Washington State Department of Natural Resources. Where published studies appear, the reference is given under study source. If the study was a contract study, the contracting laboratory responsible for conduct of the study is noted.
- 2 Any significant oncogenic effects reported in any review were noted here. Non-oncogenic chronic effects are also listed and a NOEL for systemic chronic effects is shown, if available.
- 3 Each study was evaluated for its overall usefulness in making a judgement about potential oncogenic (carcinogenic) properties. I = inadequate study; little meaningful conclusions can be drawn from study; M = minimal; although there are deficiencies in design, data evaluation and/or interpretation, the study provides useful information on oncogenic potential of chemical; additional studies would be necessary to make definitive conclusions; A = adequate; study meets currently accepted standards for design, data evaluation and interpretation. Further studies with this species are not essential to make a definitive statement about oncogenicity in this species.

Appendix H
Human Health Risk
Assessment
(Qualitative)

Section 4
Data for Analysis
of Reproductive
and Developmental
Toxicity

Developmental and Reproductive Toxicity Assessment

Two main types of toxicology tests have been used to assess the reproductive and developmental toxicity of these pesticides. Most of these tests are conducted in mouse, rat, or rabbit test populations. The attached tables give experimental details.

The potential adverse reproductive effects of pesticides have been evaluated using multigeneration exposure studies. These studies have been especially designed to evaluate the effects of compounds where a long-term, low-level human exposure pattern exists. The majority of these studies have been three generation reproduction studies. The experiment, if conducted using weanlings, uses animals (30-40 days of age, parental generation) which are randomly assigned to control or treated groups.

Exposure to test conditions starts 60 days prior to mating. The exposed males and females are mated, and litters from this mating are examined for adverse developmental effects. The offspring are sacrificed for internal examination.

A second litter from the parental generation is produced, and this litter is also examined for adverse reproductive effects. These animals are then mated as male-female within the same treatment group (brother-sister matings are avoided).

A similar pattern of examination occurs, where the first litter produced is examined both grossly and visorally, then sacrificed. The second litters produced are mated to produce the third generation.

These offspring are then also examined. Exposure to control or test conditions continues throughout the entire three-generation study.

Modifications of this general protocol are common. However, current recommendations suggest three dose levels plus a control group. The highest dose level is frequently a multiple of the human exposure level, or 10 percent of the LD_{50} . Ideally, the lowest dose tested should be a no effect level. Test protocol should allow for a minimum of 20 pregnant females per treatment group per generation.

Multigeneration reproduction studies are designed to provide data on gonadal function, estrus cycle, mating behavior, conception, implantation, abortion, fetal and embryonic development, parturition, post natal survival, lactation, maternal behavior, and post partum growth (Dixon 1986). In addition to identifying altered reproductive capability (including genetic and behavioral effects), these studies often are able to identify other systemic effects, since treatment continues throughout the animals' life.

Developmental toxicity is usually assessed using Phase II Teratology tests.

These tests are designed to assess effects on viability, growth, and birth defects. Routinely, one rodent and one non-rodent species (normally rabbits) is tested with control and two dose groups, each containing 20 rodents or 10 non-rodents. Time-mated females are treated only during the organogenesis period (days 6-15, rodents; and days 6-18, rabbits).

Doses should be selected so that the highest dose group produces some maternal toxicity. Frequently a 10 percent decrease in normal maternal body weight is used as an indication that maternal toxicity has occurred. One day prior to birth, the females are sacrificed and the fetuses are examined for viability and growth parameters and gross, visceral, and skeletal abnormalities.

Classification systems are used to order major and minor malformations dependent upon their severity and irreversibility. Minor skeletal variations such as incomplete ossification of sternum, vertebrae, or phalanges are frequently used as indicators of developmental retardation, since these effects are frequently reversible during the post natal period.

In this worst-case analysis, these endpoints will be included in identifying NOELs if these endpoints appear to be treatment related. Early and late developmental deaths are scored to determine if the test compounds may be causing lethality during gestation.

Developmental NOELs are compared to maternal NOELs to determine if the test compound causes adverse developmental effects at or below maternally toxic doses. The following ranking (score) system has been used to identify the relative developmental toxicity of test compounds:

- 1) little or no evidence of developmental toxicity in the absence of maternally toxic effects;
- 2) evidence of developmental toxicity, primarily minor abnormalities and variation observed;
- 3) evidence of developmental toxicity, including some major malformations; and
- 4) evidence of severe adverse developmental effects. Dose-related increases in major malformation in the absence of maternal toxicity.

REPRODUCTIVE TOXICITY STUDIES

Herbicide	Chemical Grade Purity	Description of Study	Doses Tested	Qualitative Ranking on Adq. Testing	Effect Level	Comments
Amitrole ^{1,6,12,13}	Technical	2-generation rats	25,100, 500,1000ppm (variable treat time)	CG=Invalid	LEL=25ppm (5mg/kg/day) NOEL=ND (50mg/kg/day)	Hyperplasia thymus No info. on exp. details Adequate #'s?
	Technical	2-generation rats (oral)	1000,5000, 25000ppm	Incomplete ¹² Inadequate ¹² CG=minimal ¹³	LOAEL=5000ppm (250mg/kg/day) NOEL=1000ppm (50mg/kg/day)	Decreased live births
Atrazine ^{1,2,6,12,13}	Unknown ¹⁴	Rats - Feed throughout gestation	50,100ppm	Unacceptable ¹² CG=Supplementary ¹³	NOEL> 100ppm 5mg/kg/day	Dietary regime altered
	Unknown ¹⁴	Rats - sub Q inj days 3,6,9	50,100,200, 800,1000. 2000mg/kg	Unknown	NOEL<50ppm	Examined pup wt and number only No teratology examination
Bromocil ^{1,6,12,13}	80% (formulated)	3-generation Rats	250ppm 12.5mg/kg/day	Unacceptable ¹² CG=minimum	NOEL>250ppm	Inadequate numbers of animals No diet analysis Inadequate histopathology Formulated versus technical grade Only one dose tested mat. tox. N.D.
	Unknown ¹⁴	Rats - sub Q inj days 3,6,9	50,100,200, 800,1000. 2000mg/kg	Unknown	LEL=800mg/kg NOEL=200mg/kg	Embryotoxicity Increased reabsorption No standard teratology evaluations done

Reproductive Toxicity Studies (continued)

2,4D	^{1,2,12,13}	97.5%	2 generation Rats	5, 20, 80 mg/kg/day	Acceptable ¹²	NOEL=5mg/kg/day (?)	Decreased mat. survival Increased fetal loss Dramatic effects in litters Adverse Reprod. effects (Data may suggest ¹² NOEL=20mg/kg/day)
Unknown			3 generation Rats	0, 100, 500, 1500ppm	Unknown	NOEL=500ppm (25mg/kg/day)	Viability, effects at high doses No adverse effects on fertility or avg. litter size at any of the doses tested
Unknown			1 + generation Rats (oral)	1000ppm	Unknown	-----	No adverse effects on reproduction *however 2 year treatment of offspring caused systemic effects of growth retardation, poor general health, diarrhea and increased mortality
Unknown			1 generation (treated only) Rats (oral)	1000, 2000ppm	Unknown	LEL=1000ppm (lowest dose tested)	Adverse effects on pup viability observed at both doses

2,4DP ^{1,13}	Acid, Technical	3 generation Rats (oral)	125,500,1000, 2000ppm	CG=Minimal ¹³	Mat. NOEL=1000ppm Mat. LEL=2000ppm Dev. NOEL=125ppm (6.25mg/kg/day)	Decreased Bulk Increase in small litters Increased postmated pup mortality
Dalapon ^{1,2,13,12}	Purity Unknown	3 generation Rats (oral)	0.03,0.1,0.3% (3000ppm) (300mg/kg/day)?	Inadequate ¹² Incomplete	NOEL> 300mg/kg/day	Insufficient data! No evidence of reproductive effect ¹² ? NOEL not established ¹² ?
	Technical	1 generation Dog (diet)	50,100,200 mg/kg/day	Incomplete ¹² Unacceptable ¹²	ND	Insufficient data! Few animals Dosing started after breeding
Dicamba ^{1,12,Z9,Z10}	Technical Banvel D (87.2% a.i.)	3 generation Rat (CD) Oral (diet) 10 males/group 20 females/group	0,50,125,250, 500ppm	Unacceptable ¹²	NOEL> 500ppm (HDT)	No adverse effects on reproduction Supplement to next study Same problems as listed below ¹² Inadequate
1,12,13,Z9,Z10	Technical DMA Salt	3 generation Rat (CD) Oral (diet)	0,50,125,250, 500 ppm	Unacceptable ¹² CG=Minimum	NOEL> 500ppm (HDT)	No adverse effects on reproduction. Problems with age, animal numbers, dose selection (no sign of tox.) short dosing prior to mating? Inadequate pathology
13,Z10	Technical	Reproduction Chickens		valid ¹³		

Reproductive Toxicity Studies (continued)

Diuron ^{1,5,13,12}	Technical 80% (Source 1)	3 generation Rat (oral)	125ppm	Unacceptable ¹² Inadequate ¹²	Reprod. NOEL> 125ppm (?) Systemic NOEL< 125ppm (?)	No adv. reprod. effect observed Body wt. decreases in F ₂ and F ₃ litters Inadequate numbers of preg. animals (8 males 16 females per group) Parental animals not necropsied, no diet analysis, no food consump. info. Single dose tested
	Technical 80% (Source 2)	3 generation Rat (oral)	125ppm	Unacceptable ¹² Inadequate ¹²	Reprod. NOEL> 125ppm(?) Sytemia NOEL> 125ppm(?)	No adv. reprod. effect observed No systemic effects observed Inadequate numbers of preg. animals (8 males 16 females per group) Parental animals not necropsied, no diet analysis, no food consump. info. Single dose tested
Fosamine ^{1,2,12}	Unformulated fosamine ammonium	One generation Rats (oral)	200, 1000, 5000/10000ppm (90 days)	Unacceptable ¹²	NOEL=5000ppm (250mg/kg/day)	Only 2 litters examined for each dose group No adverse reprod. effects observed Dose selection unjustified Minimal exp. details

Reproductive Toxicity Studies (continued)

Glyphosate ^{12,13}	Technical	3-generation Rat (CP)	0, 30, 100, 300 ppm	CG=Invalid ¹³	Unacceptable ¹³ Invalid per Canadian ¹³ revallation
1, 12, 13, Z, GRP-11, Z6		3-generation Rat (CD) Oral (diet)	0, 3, 10, 30 mg/kg/day	CG=Supplementary upgraded to Minimum ¹² Unacceptable ¹²	Reprod. NOEL=10 No adverse mg/kg/day reproductive effects observed, however Renal focal tubular dilatation observed in weanling males in F36 generation at high dose (30mg) - systemic or reproductive effect? Called reproductive ₁₂ based on 1985 review
Hexazinone ^{1,13,Z4}	Technical	3-generation Rats Oral (diet)	0, 200, 1000, 2500ppm	CG=Minimum ¹³	No differences in treated versus control groups on reproductive or lactation performance. Aug. B. Wt. of pups at weaning for F2A & F3A litters was decreased at 2500 ppm.
Picloram ^{1,2,12,Z3}	95%	3-generation (2 litter) Rat Oral (diet) 4 males 12 females	0, 0.03, 0.1, 0.3% 0 - 3000ppm 0, 15, 50, 150mg/kg	Unacceptable, ¹² non-upgradeable.	No obvious effects on reproduction however, reduced fertility at highest dose (treatment related?).

Reproductive Toxicity Studies (continued)

Picloram (con't.)

Insufficient number of animals, only 4 weeks of exposure prior to 1st mating, mating in groups inadequate number necropsied, data not clearly presented
No record of total consumption
Reduced fertility observed at 3000ppm in one generation-EPA stated ~~ngt~~ treatment related.
However, Note that NOEL I.D. by Ref 12Z and 1 was 1000ppm (50mg/kg/day)

1, 2

Fertility Mice Oral (diet) 4 days before mating 14 days after mating	0.01% 15mg/kg/day	NOEL> 15mg/kg/day	No adverse effects observed on fertility or litter size
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Simazine^{1,12,13,Z1,Z2}

Simazine 80W	3-generation Rat (CR) Oral (diet) 20 males/group 20 females/group	0, 100ppm	CG=Minimum Unacceptable with insufficient info.	Reprod. NOEL> 100ppm 5mg/kg/day	Only 1 dose tested for F2 gen. a 50ppm group added, No reprod. effects observed, some missing info. on matings, F0 not necropsied.
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Terbuthurion^{1,13}

Technical (95%)	2-generation Rat	Dose groups unknown	CG=Supplementary ¹³	Reproductive NOEL>400ppm (20mg/kg/day) Systemic NOEL> 100ppm
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Reproductive Toxicity Studies (continued)

Terbuthurion ^{1,13} (continued)	Technical	3-generation Rat	Dose groups unknown	Reproductive NOEL<400ppm Systemic NOEL=800ppm	Reproductive effects observed were decreased body wt. of weanling pups
Triclopyr ^{1,2,12,13}	Technical	3-generation Rat (SD) Oral (diet) 11-12 males 23 females	0, 3, 10, 30 mg/kg/day	Unacceptable ¹² CG=Minimum ¹³	Reproductive NOEL>30 mg/kg/day Systemic NOEL>30 mg/kg/day

DEVELOPMENTAL TOXICITY STUDIES

Herbicide	Chemical Grade	Description of Study	Doses Tested	Qualitative Ranking on Adq. Testing	Maternal Toxicity Effect Level	Developmental Toxicity Effect	Comments
Amitrole ^{1,6,12}		Teratology ³ Rats Oral	20, 100 mg/kg/day	Inadequate No visceral or skeletal exam	ND	NOEL= 100mg/kg/day	
		Teratology ¹² Rats	400, 1000 mg/kg/day	Inadequate ¹²	ND		Very limited summaries Histolog. change in thyroid at all tested doses
		Teratology ¹² Rats (CD)	100, 500, 1000 mg/kg/day	Complete ¹² Acceptable No indic. of adv. effect	NOEL= 100mg/kg/day	NOEL= 500mg/kg/day	Decreased fetal weight gain
		Teratology ⁴ Mice Oral	500, 1000, 2500, 5000 ppm comp. in water	Inadequate ¹² (no water consump. info.)	ND(?)	LOAEL= 1,000 ppm	Petotoxicity, decreased body wt., sm. fetuses under- develop. fetuses w/ immature skeletons)
Technical 91.8%		Teratology ¹² Phase II rabbits (oral)	4, 40, 400 mg/kg/day	Acceptable ¹² No data gap Possible adv. effect due to freq. and sev. of defects	NOEL= 4mg/kg/day	NOEL= 4mg/kg/day	Increased incidence of structural changes
		Teratology ⁷ Phase II Rabbits (oral)	2, 4, 8, 40 mg/kg/day	Incomplete ¹² Unacceptable ¹²	ND	NOEL> 40mg/kg	
Ansulam ¹	60% w/v						

Developmental Toxicity Studies (continued)

Ansulam (con't.)	Technical (98-99%)	Teratology Phase II rabbits (oral)	150,300, 750,1500 mg/kg/day	Incomplete ¹² Unacceptable ¹² CG=Minimum	LOEL (?) 750 mg/kg/day	NOEL (?) 300 mg/kg	Insufficient info. Many technical errors poorly conducted, confounded study
60% w/v	Teratology	8,40 mg/kg/day	Incomplete ¹² Unacceptable ¹²	ND	NOEL 40 mg/kg/day	Insufficient info. Only 2 dose levels Mat. tox. N.D.	
60% w/v	Teratology Phase II Rats (oral)	8,40	Unacceptable ¹² CG= Unacceptable ¹³	ND	NOEL 40mg/kg/day	Insufficient info. Only 2 dose levels Mat. tox. N.D.	
Technical (98-99%)	Teratology Phase II Rats (oral)	500,1000, mg/kg/day	Incomplete ¹² Unacceptable ¹² CG=Minimum	ND(?) (Insig. decrease at 1500 mg/kg/day	LOEL(?) 500mg/kg (non-stat. sig. increase in pre- implantation loss)	Insufficient info. Inadequate dose Inappropriate dosing schedule (prior to implantation) No historical control values	
Atrazine ^{1,2,6,12,13}	Technical ¹⁰	Teratology Phase II rats (oral)	10,70,700 mg/kg/day	Unacceptable ¹²	NOEL= 70mg/kg/day	NOEL< 10mg/kg/day	Increased visceral and skeletal variability
Unknown	Teratology Phase II Rats (oral)	100,500, 1000 mg/kg/day	Unacceptable ¹² CG=Minimum	NOEL 100 mg/kg/day	NOEL= 100mg/kg/day LEL= 500mg/kg/day	Fetal loss, wt. loss	

Developmental Toxicity Studies (continued)

Atrazine (con't.)	Technical	Teratology Phase II	1,5,75 mg/kg/day	Unacceptable ¹²	NOEL= 1mg/kg/day	NOEL= 5mg/kg/day	Maternal effects observed were decreased wt gain and food consumption Developmental effects observed were increased resorptions decreased fetal wt and number of fetuses
		Teratology Phase II Mouse	46mg/kg/day	Unacceptable ²	ND	NOEL= 46mg/kg/day	Used DMSO as vehicle One dose tested Insufficient info. on evaluation
Bromocil ^{1,6,12,13}	Unknown	Teratology Phase II Rat (Inhalation)	38,78, ³ 165 mg/m (=1.8,3.8, 7.9mg/kg)	Unacceptable ¹² CG=Minimum ¹³	ND	NOEL> 165mg/m ³ >7.9mg/kg	Inadequate number of animals tested No individual information No comments on visceral exam. Mat. tox. N.D. Unjustified dose selection Inadequate number of animals No comments on visceral exam or other exp. changes
2,4D ^{1,2,9,12,13}	97.5%	Teratology Phase 2 Rats (oral)	8,25,75 mg/kg/day	Incomplete ¹² CG=Minimum ¹³	NOEL> 75mg/kg/day	NOEL= 25mg/kg/day	No analysis of dosing solution Delayed ossification Fetotoxicity

Developmental Toxicity Studies (continued)

2, 4D (con't_{2,13})

Unknown	Teratology Phase 2 mg/kg (oral) gavage	75, 100, 150, 200, 250 d6-15	CG= Supplementary LEL=	¹³ NOEL= 100mg/kg/day 150mg/kg/day	ND	Evidence of develop- mental toxicity Range finding for above study. Was conducted to support dose selection in this full teratology study however, use of 75mg/kg not fully justified.
Rats (F344)						

Acid	Teratology Phase II Rats (oral)	25, 50, 100, 150mg/kg/day	Unknown	ND	NOEL=	Mat. deaths (cerebral hemorrhage at 200 and 250mg/kg/day) Skeletal abnormalities Fetotoxicity Several different sources of 2,4D tested, some inconsistency observed
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Acid	Teratology Phase II Rats (oral)	12.5, 25, 50, 75, 87.5 mg/kg/day	Unknown	ND	LEL= 12.5mg/kg/day (delayed ossifici)	Fetotoxicity Delayed ossification Skeletal abnormalities Hydrocephaly
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Acid	Teratology Mice (?)	147mg/kg (single dose)	Inadequate	ND	NOEL=147 mg/kg	Inadequate summary only available
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H-10

Developmental Toxicity Studies (continued)

2, 4D (con't.)	Acid	Teratology Phase II Hamster (oral)	20, 40, 60, 100mg/kg/day	Inadequate ²	ND	NOEL= 40mg/kg/day	Insufficient info. on abnormalities Mat. tox. N.D.
2, 4DP ^{1,13}	Acid Technical	Teratology Phase II Rats	25, 100mg/kg	CG=Guideline ¹³	ND	NOEL> 100mg/kg/(HDT)	Range finding
	Acid (94%)	Teratology Phase II Rats	10, 30, 100mg/kg	CG=Minimum ¹³	NOEL> 100mg/kg (HDT)	NOEL> 100mg/kg	Mat. tox. N.D.
	Acid Technical	Teratology Phase II Rabbits	25, 100mg/kg	CG=Minimum ¹³	NOEL= 25mg/kg (LDT)	NOEL< 25mg/kg (LDT)	Range finding Omphalocele Skeletal malformations Growth retardation
Dalapon ^{1,2,12,13}	Technical	Teratology Phase II Rats (oral)	500, 1000, 1500 mg/kg/day	Incomplete ¹² Unacceptable ¹²	NOEL= 1000mg/kg (see comments)	NOEL= 500 mg/kg/day ¹³	Decreased mat. wt. gain Some evidence of dev. tox. without mat. effect ¹² No individual data Experimental design problems Skeletal effects at all doses(?) ¹²

Developmental Toxicity Studies (continued)

Dalapon (con't.)	Unknown	Teratology Rats (oral)	250,500,1000 1500,2000 mg/kg/day	Unknown	NOEL= 500mg/kg/day	NOEL= 1500mg/kg/day	Decreases in mat. wt. gain Decrease in mat. food consumption Fetal resorptions (NS) Decreased pup wts. No teratog. effects observed
^{1,13} Dicamba ^{1,13}	Technical Acid	Teratology Rats (CD) Oral (gavage)	0,50,150, ¹³ 350,600,750 mg/kg/day d6-19	CG=Minimum	NOEL=350 mg/kg/day LEL=600 mg/kg/day	See next study	Pilot study Mat. tox. included behavioral reactions and gross stomach lesions.
^{1,13} 1,Z10,13	Technical Acid	Teratology Rats (CD) Oral (gavage) 20-24 rats/gr	0,64,160, ¹² 400 mg/kg/day d6-19	Acceptable ¹³ CG=Minimum Inadequate (see comments)	NOEL= 160mg/kg/day LEL=400 mg/kg/day	NOEL<64 mg/kg/day LEL=64 mg/kg/day	Mat. tox. observed including ataxia, salivation decreased motor activ., mortality and decreased body wts. and food consumption. Problems with study since skeletal malf. were observed in all groups (non dose- related) however, incidence of misshapen inter- parietal, occipital and parietal skeletal bones only in treated groups was suggestive and resulted in a LEL=64mg/kg/day (lowest dose tested).

Developmental Toxicity Studies (continued)

Dicamba (con't.)	Unknown	Teratology Rabbits Oral (gavage) 21-22/group	0, 1, 3, 10 mg/kg/day	Inadequate ¹² Unacceptable ¹² CG= Supplementary ¹³	ND	?NOEL=3.0 mg/kg/day	No individual data etc Supplement to above observed study due to disease and mortality Difficult to identify treatment related effects. Possible effects on male/ female ratio and fetal body wt. on 10mg/kg/day group.
							Inadequate numbers of preg., combined this study with repeat below. No individual animal data given. No teratogenic effect observed.
Bonnel Technical (87.7%)		Teratology Rabbits Oral (gavage) 31-35/group	0, 1, 3, 10 mg/kg/day d6-18	Unacceptable ¹² Inadequate ¹² CG= Supplementary ¹³	ND		
Technical		Teratology Rabbits (NZ) Oral (gavage) 10/group	0, 0.5, 1, 3, 10, 20 d6-18	CG= Supple- mentary ¹³ , Z10	NOEL 10mg/kg/day	NOEL= 0.5mg/kg/day LEL= 1.0mg/kg/day	Mat. toxicity observed included reduced wt. gain and decreased activity. Data on skeletal and soft tissues not given. Too few animals (10/group) Increased fetal resorbptions noted in in 1.0mg/kg/day group

Developmental Toxicity Studies (continued)

Diuron ^{1,5,13,12}	Karmex (80% Diuron)	Teratology Phase II	125, 250, 500	Unacceptable ¹² CG=	NOEL= 250mg/kg/day ¹³	LEL= 125mg/kg/day (lowest dose tested)	Insufficient Fetal wt. decreased Wavy ribs Mat. wt. reduced Dev. NOEL not determined Stat. Sig. Delayed ossification in lowest dose tested No individual data given
		Rat (oral)	mg/kg/day	Supplementary			
Fosamine ^{1,2,12}	Krenite	Teratology Phase II	215mg/kg	Unacceptable ¹²	NOEL=215 mg/kg		No adv. develop. tox. observed at 215mg/kg No study details Tabular summary only Totally unacceptable
		Mouse (?)					
Fosamine ^{1,2,12}	Krenite	Teratology Phase II	200, 1000, 10000ppm	Unacceptable ¹²	ND	NOEL= 1000ppm(?) (21mg/kg/day a.i.)	Mat. tox. N.D. Stat. Sig. Hydronephrosis noted at high dose (P=.04) Minimal exp. details available Dose selection not justified Test material not described No individual data available
		Rats (oral) (28 females/ group)	(equiv. to 207mg/kg/day a.i.)				

Developmental Toxicity Studies (continued)

Glyphosate ^{1,2,12,13,27}	Technical 98.7%	Teratology Rat (CD) Oral (gavage) 25/group	0, 300, 1000, 3500 mg/kg/day	Complete ¹² Acceptable ¹³ CG=Minimum	NOEL=1000 mg/kg/day LEL=3500 mg/kg/day	Mat. toxicity observed included inactivity, death, stomach hemorrhages, decreased wt. gain. Developmental toxicity observed was delayed ossification (high dose) and structural malformations (high dose - single litter) Developmental toxicity only observed at doses causing significant maternal toxicity.
1,2,12,13,28	Technical 98.7%	Teratology Rabbit Oral (gavage) @6-27 (16/group)	0, 75, 175, 350 mg/kg/day	Complete ¹² Acceptable ¹³ CG=Minimum	NOEL=175 mg/kg/day LEL=350 mg/kg/day	Signs of mat. tox. observed at 350 mg/kg/day included death, soft stools, diarrhea, nasal discharge. Note: major structural malform- ations were observed in 2 fetuses in 175 mg/kg/day group and 1 fetus in 350 mg/kg/day group. Were not stat. sig. and were not considered to be related to treatment. For worst case analysis, will set developmental tox. NOEL at 75 ² mg/kg/day.

Developmental Toxicity Studies (continued)

Glyphosate (con't.)	12,13	Technical	Teratology Rabbit d6-18	10,30 mg/kg	Invalid ¹² CG=Invalid ¹³ (IBT)		
	13	Technical	Teratology Rabbit		CG=Invalid ¹³ (IBT)		
Hexazinone	1,2,Z4	Technical	Teratology Rat Oral (diet) 25-27 rats/grp. d6-15	0,200,1000, 5000ppm		NOEL=1000ppm	NOEL>5000ppm
							Mat. effects observed at 5000ppm included decreased food consumption and body wt. No adverse developmental effects were observed at any test doses.
1,13,Z4		Technical	Teratology Rabbit Oral (gavage) d6-19	0,20,50,125 mg/kg/day	CG= Minimum ¹³	NOEL>125 mg/kg/day	NOEL=50 mg/kg/day
							No maternal effects. The highest dose level showed a higher percentage (16.2%) of fetuses showing skeletal variants than controls in skeletal development. These differences included delayed ossification in extremities and extra ribs.
1,Z5			Teratology Rabbit				NOEL=20
							No exp. details known

Developmental Toxicity Studies (continued)

Picloram ^{1,2,23,12}	No purity stated 35/group d6-15	Teratology Rat (SD) Oral (gavage)	0, 500, 750, 1000 mg/kg/day	CG= Supplemental ¹² Unacceptable ¹² Upgradeable ¹²	NOEL=500 mg/kg	NOEL<500 ²³ mg/kg/day LOAEL=500 mg/kg/day	No NOEL for develop. tox. determined. Mat. tox. observed. Ref 12 - Unacceptable due to lack of purity info., no analysis of dosing solution, poor copies of individual data. Decreased viability index at highest dose. Presence of minor variations and no sig. major malf. Treatment related?
<hr/>							
12		Teratology Rabbit Oral (gavage) d6-18 18-23/group	0, 40, 200, 400mg/kg	Unacceptable ¹² Upgradable ¹²	NOEL=40mg/kg	NOEL>400 mg/kg(?)	No teratogenic effects observed. ¹² Insufficient info. to evaluate (no ¹² individual data).
<hr/>							
Simazine ^{1,13,12}	Technical 97%	Teratology Rabbit Oral (gavage) 18/group	0, 5, 75, 200 mg/kg	CG= Supplementary, Guideline Acceptable with no adverse ¹² effect	NOEL=5mg/kg	NOEL=75 ¹³ mg/kg/day NOEL=5 ¹² mg/kg/day	Maternal effects observed at 75mg/kg included tumors, abortions, & decreased body wt. gain and food consumption. Decreased fetal wt. and increased skeletal variations at 200mg/kg. Late resorptions observed at ¹² 75 and 200mg/kg.

Developmental Toxicity Studies (continued)

Terbuthiuron ^{1,13}	Technical	Teratology Rabbit	Unknown	NOEL>25mg/kg	Minimal details known ; doses, route, of exposure, etc. Maternal tox.? Not given.
	1,13	Technical	Teratology Rat	Highest dose 1800ppm	Minimal details known doses, route, of exposure, etc. Maternal tox.? Not given.
	1	Unknown	Teratology Rat Dermal	NOEL=237 mg/kg/day	No experimental details known.
Triclopyr ^{1,2,12,13}	Technical (98.5%)	Teratology Rat (SD) Oral (gavage) 25/group	0,50,100, 200 mg/kg/day d6-15	Acceptable ¹² CG=Minimal ¹³	Maternal toxicity observed was change in wt. gain, food consumption. Fetal effects (delayed ossification) at 200mg/kg/day were ascribed to maternal effects.
	Technical (>95%) Oral (gavage)	Teratology Rabbit d6-18 15/test groups 25/control groups	0,25,50,100 mg/kg/day	Unacceptable ¹² CG=Supplementary ¹²	Heavy mortality at all doses and among controls. Attributed by investigators to volume administered by gavage. Insufficient info. to evaluate

Triclopyr (con't.) Technical
(Dow W 233)

Teratology
Rabbit
Intubation
20/group

Unacceptable¹²
CG=Minimum

0,10,25
mg/kg
d6-18

NOEL=?

NOEL < 10
mg/kg/day

Enteritis deaths in all groups.
Treatment related effects at 25mg/kg in dams? No other signs of mat. tox.
Minor anomalies were increased (not sig.) above control values.

References for Developmental and Reproductive Toxicity

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2. A.M. Shipp, M.L. Hogg, K.S. Crump, and R.L. Kodell. Worst case analysis study on forest plantation herbicide use. Forest Land Management Division, Department of Natural Resources, State of Washington, May 1986.
3. T.B. Gaines, R.D. Kimbough, and R.E. Linder. The toxicity of amitrole in rats. *Tox. Apl. Pharm.* 26:118-129 (1973).
4. H. Tjalve. Fetal uptake and embryotoxic effects of amino triazole in mice. *Arch. of Tox.* 33:41-48 (1974).
5. EPA, 1985a (EIS pg 1-32).
6. J.L. Shardein. Chemically induced birth defects. *Drug and Chemical Toxicology* Vol. 2, Marcel Dekker, Inc., New York 1985 pp. 577-617.
7. EPA, 1984b.
8. EPA, 1984c.
9. Ciba Geigy, 1985.
10. EPA, 1984c.
11. Hayes, 1982 EPA stated study evaluation.
12. Medical Toxicology Reviews. California Department of Food and Agriculture Reports, Pest Management, Environmental Protection and Worker Safety, State of California, February 1987.
13. Tox 1-liners. We have not adjusted any of the doses for percent purity of compound tested.
14. Peters and Cook (from Crump pg. 161).
15. EPA Dicamba Registration Standard (6/11/85?)(SC-8).

Appendix H
Human Health Risk
Assessment
(Qualitative)

Section 5
Data for Analysis
of Immunotoxicity
and Neurotoxicity

Neurotoxicity of Herbicides

1. Summary of data

For 10 of the herbicides studied (amitrole, bromacil, 2,4-DP, dalapon, dicamba, diuron, glyphosate, hexazinone, picloram, triclopyr), there is no reported evidence of any sign of toxicity involving the central and/or peripheral nervous system.

For atrazine, fosamine, simazine, and tebuthiuron, signs indicative of a nervous system involvement were reported for doses equivalent to their LD50s.

For simazine, nervous symptoms were also observed following 21-day exposure (1,000-2,000 mg/kg) in rabbits; for atrazine following 6-month exposure in dogs (1,500 ppm) and after 3-month exposures in rats (75 mg/kg); and for asulam following 5-day exposure of dogs to 2,000 mg/kg.

2,4-D caused no neuropathy in animals, but caused brain lesions and EEG alterations following exposure to 100-300 mg/kg. Peripheral neuropathy and brain lesions have been observed in humans exposed to high or lethal doses. A decreased nerve conduction velocity has been found in workers chronically exposed to 2,4-D and 2,4,5-T.

2. Evaluation

With the exception of the delayed neurotoxicity test required for all new organophosphates, no other test for central or peripheral nervous system toxicity is required for registration of pesticides. Therefore, specific tests aimed at detecting neurotoxicity are not normally done with compounds such as the herbicides under consideration. However, any sign of toxicity, including those suggestive of a nervous system involvement, are usually recorded and reported during acute, subchronic, and chronic studies. No such indications were reported for 10 out of 16 herbicides, suggesting that no specific signs were observed.

For five additional herbicides, certain signs suggestive of a possible nervous system involvement were reported; however, these occurred mostly when lethal or very high doses were administered. Atrazine provided some indications of neurotoxicity at doses well below its lethal dose; although the doses are still quite high and only two animal studies are available, this potential effect should be kept in consideration.

2,4-D has caused peripheral neuropathy and brain lesions in humans exposed to very high doses (accidental poisoning or suicide).

One study reported a decrease in nerve conduction velocity in chronically 2,4-D exposed workers. However, there was a concomitant exposure to 2,4,5-T (and most probably dioxins), which makes it impossible to ascribe this effect to 2,4-D.

Neuropathy has not been observed in laboratory animals, but exposure of mice and rats to doses as low as 100 mg/kg of 2,4-D were found to cause brain lesions in one study. No determination of NOEL is possible from the available studies. 2,4-D should be reported as a potentially neurotoxic compound, possibly only in a few susceptible individuals and only at high doses.

Immunotoxicity of Herbicides

1. Summary of Data

There is no reported evidence of immunotoxicity for 13 herbicides (amitrole, asulam, bromacil, 2,4-DP, dalapon, dicamba, fosamine, glyphosate, hexazinone, picloram, simazine, tebuthiuron and triclopyr). Diuron (250 mg/kg in the diet) increased spleen weights, and atrazine (100 mg/kg in the diet) caused lymphopenia (decreased white cells count). 2,4-D had some immunotoxic effects at high doses (200 mg/kg and up), and a breakdown product of 2,4-D depressed cell-mediated immunity in rats at the dosage of 30 ppm.

2. Evaluation

No specific tests for immunotoxicity are required for pesticide registration, and therefore no experiments aiming at detecting alterations in immune function are performed. Some evidence of toxicity to the thymus or the spleen could surface during autopsies following subchronic and/or chronic studies, but these have not been reported for the herbicides under consideration.

Atrazine-caused lymphopenia at a dose level (100 mg/kg) similar to that which caused some evidence of neurotoxicity. Although the information available is limited, exposure levels should be kept below these values.

Acute administration of 2,4-D caused alteration of cell-mediated responses at 200 mg/kg and of humoral responses at 500 mg/kg, when overt clinical manifestations of toxicity and histopathological alterations in brain were also present. No effects were found following repeated exposures. Exposure to such high doses of 2,4-D are unlikely to occur. Exposure to 2,4-dinitrophenol—a breakdown product of 2,4-D—alters cell-mediated immunity at doses of 30 ppm. Safe exposure levels should be kept below these values.

Data for Analysis of Immunotoxicity and Neurotoxicity

1. Amitrole:	NEUROTOX.	: No reported evidence of neurotoxicity in subchronic studies.
	IMMUNOTOX.	: No reported evidence of immunotoxicity in acute and subchronic studies.
2. Asulam:	NEUROTOX	: Vomiting and anorexia in dogs given 2,000 mg/kg/day for 5 days (only dose tested).
	IMMUNOTOX.	: No reported evidence of immunotoxicity in acute and subchronic studies.
3. Atrazine:	NEUROTOX.	: Tremor, ataxia, hypoactivity in rats after LD ₅₀ doses. Rear limb muscular tremors in dogs following 6 months feeding of 1,500 ppm. Impairment of learning and alteration in EEG activity in rats given 1/40 of the LD ₅₀ (i.e. 75 mg/kg/day) of Toxurazine (15% atrazine, 15% chlorinol, 30% aminotriazole) for 3 months. (Desi et al., Acta Physiol. Acad. Scient. Hung. 60:-1-8, 1982).
	IMMUNOTOX	: 100 mg/kg in diet caused lymphopenia in rats (Vos et al. In Pesticide Chemistry. Human Welfare and the Environment, Vol. 3, p. 497-506, 1983).

Summary of Neurotoxicity and Immunotoxicity of Herbicides

- | | | |
|-------------|------------|--|
| 4. Bromacil | NEUROTOX. | : No reported evidence of neurotoxicity in acute and subchronic studies. |
| | IMMUNOTOX. | : No reported evidence of neurotoxicity in acute and subchronic studies. |
| 5. 2,4-D | NEUROTOX. | : A few reports in humans of neuropathies (Goldstein et al. JAMA 171, 1306, 1959; Monarca and Di Vito, Folia Medica (Naples) 44, 480, 1962; Todd, J. Iowa Med. Soc. 52, 663, 1962; Berkley and Magee, Arch. Int. Med. 111, 351, 1963). |
- Decreased conduction velocity in sural nerve in workers chronically exposed to 2,4-D and 2,4,5,-T (Singer et al., Env. Res. 29, 297, 1982).
- Memory impairment and polyneuritis in patient following ingestion of 300 mg/kg (Brandt, Ugeskr. Laeg. 133, 500, 1971).
- Brain lesions reported in 2 humans who committed suicide with 2,4-D (Nielson et al. Acta Pharmacol. Toxicol. 22, 226, 1965; Dudley and Thapar, Arch. Pathol. 94, 270, 1972).
- No neuropathy seen in rats, chickens, pigs, treated either orally or dermally for up to one year (Desi et al., Arch. Env. Health, 4, 95, 1962; Bjorklund and Erne, Acta Vet. Scand. 7, 364, 1966; Mattson et

al. *Fund. Appl. Toxicol.* 6, 175, 1986; *Neurobehav. Toxicol. Teratol.* 8, 255, 1986).

No brain lesions in rats fed 500 ppm for 2 years (Hansen et al. *Toxicol. Appl. Pharmacol.* 20, 122, 1971).

EEG alternations in rats following acute and chronic treatment (200 mg/kg) (Desi and Sos, *Med. Acad. Sci. Hung* 18, 429, 1962); Desi et al. *Arch. Env. Health* 4, 95, 1962).

Acute doses (200 mg/kg and up) cause myotonia in rats and dogs (Brody, *Arch. Neurol.* 28, 243, 1973; Drill and Hiratzka, *Arch. Ind. Hyg. Occup. Med* 7, 61, 1953).

Previous exposure to 250 mg/kg increases concentration of [¹⁴C] 2,4-D in brain by sevenfold and in other tissues by two-three fold (Elo and Ylitalo, *Toxicol. Appl. Pharmacol.* 51, 439, 1979).

Acute dermal dose of 500 mg/kg or subacute (3 weeks) dermal doses of 100-300 mg/kg in mice cause histopathological lesions in CNS including perivascular edema and ganglial cells necrosis (Blakley and Shiefer; *J. Appl. Toxicol.* 6, 291, 1986).

IMMUNOTOX. : Altered immune functions in rats following 3 months

exposure to 30 and 300 ppm 2,4-dichlorophenol, breakdown product of 2,4-D. (Exon et al., J. Toxicol. Ev. Health 14, 723, 1984).

Exposure in utero to 200 mg/kg reduces lymphocyte mitogen responsiveness in 6 week-old offspring (subtle injury to lymphocyte precursors?). No changes in humoral immunity. (Blakley and Blakley, Teratology 33, 15, 1986).

Acute dermal dose of 200 and 500 mg/kg in mice suppressed antibody production against sheep RBC but not the proliferative responses induced by other mitogens. Subacute dermal exposure to 100-300 mg/kg had no effect on these parameters (Blakley and Schiefer, J. Appl. Toxicol. 6, 291, 1986).

6. 2,4-DP	NEUROTOX.	: No reported evidence of neurotoxicity in acute and subchronic studies.
	IMMUNOTOX.	: No reported evidence of immunotoxicity in acute and subchronic studies.
7. Dalapon	NEUROTOX.	: No reported neurotoxicity in acute and subchronic studies.
	IMMUNOTOX.	: No reported evidence of immunotoxicity in acute and subchronic studies.

8. Dicamba	NEUROTOX	: Sciatic nerve damage observed in hens at LD50 dose.
	IMMUNOTOX	: No reported evidence of immunotoxicity in acute and subchronic studies.
9. Diuron	NEUROTOX	: No reported evidence of neurotoxicity in acute and subchronic studies.
	IMMUNOTOX	: 250 mg/kg in diet increased spleen weight in rats (Vos et al. IN Pesticide Chemistry. Human Welfare and the Environment, Vol. 3, p. 497-506, 1983).
10. Fosamine	NEUROTOX	: Tremors and convulsions at LD ₅₀ dosage.
	IMMUNOTOX	: No reported evidence of immunotoxicity in acute and subchronic studies.
11. Glyphosate	NEUROTOX	: No reported evidence of neurotoxicity in acute and subchronic studies.
	IMMUNOTOX	: No reported evidence of immunotoxicity in acute and subchronic studies.
12. Hexazinone	NEUROTOX	: No reported evidence of neurotoxicity in acute and subchronic studies.
	IMMUNOTOX	: No reported evidence of immunotoxicity in acute and subchronic studies.
13. Picloram	NEUROTOX	: No reported evidence of neurotoxicity in acute and subchronic studies.

	IMMUNOTOX	: No reported evidence of immunotoxicity in acute and subchronic studies.
14. Simazine	NEUROTOX	: 1,000 and 2,000 mg/kg to rabbits in 21-day dermal exposure caused uncoordination, paralysis, and decreased brain weight.
		LD ₅₀ dose in rabbit caused paralysis, tremor, convulsions.
		LD ₅₀ dose in rat caused hypoactivity, muscular weakness, labored breathing, convulsions, ataxia.
	IMMUNOTOX	: No reported evidence of immunotoxicity in acute and subchronic studies.
15. Tebuthiuron	NEUROTOX	: At LD ₅₀ doses in mice, rat, cat, and dog caused hyper-irritability, loss of sighting reflex, ataxia, emesis, tremors, convulsions.
	IMMUNOTOX	: No reported evidence of immunotoxicity in acute and subchronic studies.
16. Triclopyr	NEUROTOX	: No reported evidence of neurotoxicity in acute and subchronic studies.
	IMMUNOTOX	: No reported evidence of immunotoxicity in acute and subchronic studies.

Appendix H
Human Health Risk
Assessment
(Qualitative)

Section 6
Data for Evaluation
of Human Epidemiology

I. Cohort Studies

The following eight cohort studies look at cancer deaths and incidence among groups of workers exposed to phenoxy acids. One also evaluates a group exposed to Amitrole. These studies will be referred to throughout this section as they pertain to the specific cancer being evaluated. They are presented here in more detail.

The studies are ordered according to the total number of deaths or cases observed. These studies include fairly small cohorts, and some rarer diseases may not be represented in their findings.

All the studies except that by Lynge (1985) look only at mortality. Lynge used information on the incidence of cancer in his cohort.

Cohort Studies

Lynge 1985 Denmark Manufacturer Cohort Total observed cancer incidence = 208 among 200 individuals	4,459 workers at two factories in Denmark. 3,390 males and 1,069 females 940 worked in mfg. & pkg. of phenoxy herbicides 1,226 worked in manual service functions 1,667 worked in mfg. & pkg. of other chemicals Remainder worked office and unspecified jobs 59% males and 50% females worked less than 1 year. Study reports results by department and for the entire cohort.
Zack & Gaffey 1983 USA Manufacturer Cohort Total observed deaths = 163	884 white male hourly workers at Monsanto in Nitro, West Virginia employed at least one year. Exposure to 2,4,5-T was determined only for deceased. Study reports SMR's for the entire cohort and PMR's for the deceased by exposed and unexposed. Only 58 of the 163 deaths were 2,4,5-T exposed.
Riihimaki et al. 1982 Finland Herbicide Applicators Total observed deaths = 144	1,971 male herbicide applicators of four employers with at least two weeks exposure to 2,4-D or 2,4,5-T 75% worked less than eight weeks total.
Axelson et al. 1980 Sweden Railroad Sprayers Total observed deaths = 45	348 RR herbicide sprayers with exposure > 45 days Study reports SMR's for the total cohort and three distinct subgroups: phenoxy acid, amitrol and combined exposure.

Zack & Suskind 1980
USA
Manufacturer Cohort
Total observed
deaths = 32

121 white male workers with chloracne from 1949
accident at Monsanto in Nitro, West Virginia.

Thiess et al. 1982
Germany
Manufacturer Accident
Total observed
deaths = 21

74 workers at a BASF AG plant in Germany involved
in a 1953 accident—66 with chloracne.

Ott et al. 1980
USA
Manufacturer Cohort
Total observed
deaths = 11

204 male workers at Dow Chemical who worked in a
2,4,5-T area for at least one month—157 worked
less than one year.

Cook et al. 1980
USA
Manufacturer Cohort
Total observed
deaths = 4

61 male workers at Dow Chemical in a 1964 incident.
49 developed chloracne.

II. Case Control Studies

Various case-control studies have been conducted to look at risk factors for specific cancers. All the case control studies reported in this section were specifically concerned about associations between phenoxy acid exposures and the cancer being studied.

The case control studies are summarized under the headings of the specific cancers being evaluated.

III. Presentation of Epidemiology Studies by Type of Cancer

Summaries of all cohort studies and relevant case-control studies are presented below for several types of cancers. Risk ratios are presented when available.

All the cohort studies have been included for each cancer to provide completeness. In some cases, several small cohort studies, with little information on their own, combine to present possible patterns of disease.

Patterns of risk for cancer, not only "proof" of a carcinogenic effect, should be considered in worst case analyses.

Overall Cancers

Toxicity/Study	Point Estimate of Risk		Comment
	≥1.0	≤1.0	
Cohort Studies			
Lynge 1985 Manufacturer Cohort	1.05	0.99 0.88	Based on all 159 male cases. Based on all 49 female cases.
		0.87	Based on 28 male cases in phenoxy acid manufacture and packaging. Based on 13 female cases in phenoxy acid manufacture and packaging.
Zack & Gaffey 1983 Manufacturer Cohort	1.13	0.82	Based on all 35 male deaths. PRM based on 9 male deaths among 2,4,5-T exposed workers.
Riihimaki et al. 1982 Herbicide Applicators		0.71	Based on all 26 male deaths.
		0.82	Based on 20 male deaths with ten year latency period.
Axelson et al. 1980 Railroad Sprayers	1.4		Based on all 17 male deaths.
	1.1 (1.9)		Based on 6 male deaths with only phenoxy acid exposure. (with a ten year latency period)
	2.1 (3.4*)		Based on 6 male deaths with phenoxy acid and amitrol exposure. (with a ten year latency period)
	1.5 (1.5)		Based on 5 male deaths with only amitrol exposure. (with a ten year latency period)
Zack & Suskind 1980 Manufacturer Accident		1.0	Based on all 9 male deaths.
Thiess et al.	1.7		Based on all 7 male deaths (two 1982 cancer patients still alive). Manufacturer Accident
Ott et al. 1980 Manufacturer Cohort		0.28	Based on the 1 male death.
Cook et al. 1980 Manufacturer Cohort	2.0		Based on all 3 male deaths.

* = Significant at the $p=0.05$ level.

Lung Cancer

Toxicity/Study	Point Estimate of Risk		Comment
	≥1.0	≤1.0	
Cohort Studies			
Lynge 1985 Manufacturer Cohort	1.19 2.21 2.06*		Based on 38 male cases. Based on 6 female cases. Based on 11 male cases in phenoxy acid manufacture and packaging. Based on 1 female case in phenoxy acid manufacture and packaging.
Zack & Gaffey 1983 Manufacturer Cohort	1.28 1.41 1.68		Based on 14 male deaths. PRM based on 6 male deaths among 2,4,5-T exposed workers.
Riihimaki et al. 1982 Herbicide Applicators	1.1		Based on 12 male deaths with ten year latency period.
Axelson et al. 1980 Railroad Sprayers	1.4	0.0	Based on 3 male deaths. Based on 0 male deaths with only phe- noxy acid exposure.
	1.9 (2.9)		Based on 1 male death with phenoxy acid and amitrol exposure. (with a ten year latency period)
	3.2 (2.6)		Based on 2 male deaths with only amitrol exposure. (with a ten year latency period)
Zack & Suskind 1980 Manufacturer Accident	1.66		Based on 5 male deaths.
Thiess et al. 1982 Manufacturer Accident	2.3 (2.9) (4.6**)		Based on all 7 male deaths (one lung cancer patient still alive). (ten & 20 year latency periods)
Ott et al. 1980 Manufacturer Cohort	+		Based on 1 male death.
Cook et al. 1980 Manufacturer Cohort		0.0	Based on 0 male deaths.

* = Significant at the $p=0.05$ level.** = Significant at the $p=0.07$ level.

Stomach Cancer

Toxicity/Study	Point Estimate of Risk		Comment
	≥1.0	≤1.0	
Cohort Studies			
Lynge 1985 Manufacturer Cohort	1.29 1.36	0.68 0.0	Based on 12 male cases. Based on 1 female case. Based on 2 male cases in phenoxy acid manufacture and packaging. Based on 0 female cases in phenoxy acid manufacture and packaging.
Zack & Gaffey 1983 Manufacturer Cohort		0.63 0.0	Based on 1 male death. PRM based on 0 male deaths among 2,4,5-T exposed workers.
Riihimaki et al. 1982 Herbicide Applicators	1.1		Based on 4 male deaths with ten year latency period.
Axelson et al. 1980 Railroad Sprayers	2.2 3.1 (6.1*) 3.1 (5.6)		Based on 3 male deaths. Based on 2 male deaths with only phenoxy acid exposure. (with a ten year latency period) Based on 1 male death with phenoxy acid and amitrol exposure. (with a ten year latency period)
		0.0	Based on 0 male deaths with only amitrol exposure.
Zack & Suskind 1980 Manufacturer Accident		0.0	Based on 0 male deaths.
Thiess et al. 1982 Manufacturer Accident	4.7* (5.8*) (8.7*)		Based on all 3 male deaths. (with a ten year latency period) (with a twenty year latency period)
Ott et al. 1980 Manufacturer Cohort		0.0	Based on 0 male deaths.
Cook et al. 1980 Manufacturer Cohort		0.0	Based on 0 male deaths.

* = Significant at the p=0.05 level.

Leukemia

Toxicity/Study	Point Estimate of Risk		Comment
	≥1.0	≤1.0	
Cohort Studies			
Lynge 1985	1.11		Based on 5 male cases.
Manufacturer Cohort	2.08		Based on 2 female cases.
	1.35		Based on 1 male case in phenoxy acid manufacture and packaging.
	4.0		Based on 1 female case in phenoxy acid manufacture and packaging.
Riihimaki et al. 1982		0.0	Based on 0 male deaths with ten year latency period.
Herbicide Applicators			
Axelson et al. 1980 Railroad Sprayers	+		Based on 2 male deaths.
	+		Based on 1 male death with only phenoxy acid exposure.
	(+)		(with a ten year latency period)
	+		Based on 1 male death with phenoxy acid and amitrol exposure.
	(+)		(with a ten year latency period)
Zack & Suskind 1980	+		Based on 2 male deaths.
Manufacturer Accident			
Thiess et al. 1982		0.0	Based on 0 male deaths.
Manufacturer Accident			
Ott et al. 1980		0.0	Based on 0 male deaths.
Manufacturer Cohort			
Cook et al. 1980		0.0	Based on 0 male deaths.
Manufacturer Cohort			

* = Significant at the $p=0.05$ level.

+ = Unquantified excess risk.

Hodgkin's

Toxicity/Study	Point Estimate of Risk		Comment
	≥1.0	≤1.0	
Case-Control Studies Hardell, Eriksson et al. 1981	4.8*		169 cases of malignant lymphoma (including 60 Hodgkins lymphomas) and 338 controls. Controls were from the general population. Cases and controls with exposure to chlorophenol were excluded. A dose response was observed. SMR is reported for the entire cohort—it was reported that there was no observable difference in risk between the Hodgkin's and non-Hodgkin's cases.
Hardell	5.5*		169 malignant lymphoma cases (including 60 Hodgkins lymphomas and 154 controls.) This is the same case population as above (Hardell & Eriksson 1981), but new controls were chosen from males with colon cancer. A dose response observed. SMR is reported for the entire cohort.
Hardell & Bengtsson 1983	5.0*		60 cases and 335 controls. Controls were from the general population. Cases & controls with high exposure to chlorophenols were excluded. This is a refinement of the two studies presented above (Hardell 1981/Hardell & Bengtsson 1983)
Hoar, Blair et al. 1986		0.9	71 cases and 984 controls. Controls were from the general population. Exposure was based on reported herbicide use.

* = Significant at the $p=0.05$ level.

Hodgkin's

Toxicity/Study	Point Estimate of Risk		Comment
	≥ 1.0	≤ 1.0	
Cohort Studies Riihimaki et al. 1982		0.0	Based on 0 male deaths.
Axelsson et al. 1980	+		Based on 2 male deaths.
	+		
	(+)		Based on 1 male death with only phenoxy acid exposure (with ten year latency period).
	0.0		Based on 0 male deaths with phenoxy acid & amitrol exposure
	+		
	(+)		Based on 1 male death with only amitrol exposure (with ten year latency period).
Zack & Suskind 1980	+		Based on 1 male death.
Thiess et al. 1982		0.0	Based on 0 male deaths.
Ott et al. 1980		0.0	Based on 0 male deaths.
Cook et al. 1980		0.0	Based on 0 male deaths.

+ = Unquantified excess risk.

Non-Hodgkin's

Toxicity/Study	Point Estimate of Risk		Comment
	≥1.0	≤1.0	
Cohort Studies			
Hardell 1979			Pilot study of 17 cases. 14 cases had employment consistent with exposure (farming, forestry, sawmill, painting, and building).
Case-Control Studies			
Hardell, Eriksson et al. 1981	4.8*		169 cases of malignant lymphoma (including 109 non-Hodgkins lymphomas) and 338 controls. Controls were from the general population. Cases and controls with exposure to chlorophenol were excluded. A dose response was observed. SMR is reported for the entire group - it was reported that there was no observable difference in risk between the Hodgkin's and non-Hodgkin's cases.
Hardell 1981	5.5*		169 malignant lymphoma cases (including 109 non-Hodgkins lymphomas) and 154 controls. This is the same case population as above (Hardell & Eriksson 1981), but new controls were chosen from males with colon cancer. A dose response observed. SMR is reported for the entire cohort.
Hoar, Blair et al. 1986	1.6 (6.0*)		170 cases and 948 controls. Controls were from the general population. Exposure was based on reported herbicide use. (SMR increased to a significant 6.0 for herbicide use of at least 20 times per year). A dose response was demonstrated.
Pearce, Smith et al. 1986	1.4		83 cases matched with two sets of controls—168 controls with other cancers and 228 controls from the general population.

* = Significant at the $p=0.05$ level.

Non-Hodgkin's

Toxicity/Study	Point Estimate of Risk		Comment
	≥1.0	≤1.0	
Cohort Studies			
Riihimaki et al. 1982		0.0	Based on 0 male deaths.
Axelsson et al. 1980		0.0	Based on 0 male deaths.
Zack & Suskind 1980		0.0	Based on 0 male deaths.
Thiess et al. 1982		0.0	Based on 0 male deaths.
Ott et al. 1980		0.0	Based on 0 male deaths.
Cook et al. 1980		0.0	Based on 0 male deaths.

Soft Tissue Sarcoma (STS)

Toxicity/Study	Point Estimate of Risk		Comment
	≥1.0	≤1.0	
Case-Control Studies			
Hardell & Sandstrom 1979	5.3*		46 cases and 201 controls. Controls were from the general population. Cases & controls with chlorophenol exposures were excluded.
Eriksson, Hardell et al. 1981	6.8*		110 cases & 220 controls. Controls were from the general population. Cases & controls with chlorophenol exposures were excluded.
Smith, Pearce et al. 1984	1.3		82 cases and 92 controls. Controls were selected from males with other cancers. Case & controls with chlorophenol exposures were excluded.
Hoar, Blair et al. 1986	0.9		71 cases and 948 controls. Controls were from the general population. Exposure was based on reported herbicide use.

* = Significant at the $p=0.05$ level.

Soft Tissue Sarcoma (STS)

Toxicity/Study	Point Estimate of Risk		Comment
	≥1.0	≤1.0	
Cohort Studies			
Lynge 1985	2.72	0.0	Based on 5 male cases. Based on 0 female cases.
	3.33	0.0	Based on 1 male case in phenoxy acid manufacturing and packing. Based on 0 female cases in phenoxy acid manufacturing and packing.
	5.19*	0.0	Based on 3 male cases in manual services. Based on 0 female cases in manual services.
	1.38	0.0	Based on 1 male case in other chemical manufacturing and packing. Based on 0 female cases in other chemical manufacturing and packing.
Zack & Gaffey 1983	+		Based on 1 male death in plant.
	+		PMR based on 1 male death to a 2,4,5-T exposed worker.
Riihimaki et al. 1982		0.0	Based on 0 male deaths.
Axelsson et al. 1980	+	0.0	Based on 1 male death.
		0.0	Based on 0 male deaths with only phe- noxy acid exposure.
		0.0	Based on 0 male deaths with phenoxy acid & amitrol exposure.
	+		Based on 1 male death with
	(+)		only amitrol exposure (with ten year latency period).
Zack & Suskind 1980	+		Based on 1 male death.
Thiess et al. 1982		0.0	Based on 0 male deaths
Ott et al. 1980		0.0	Based on 0 male deaths.
Cook et al. 1980	+		Based on 1 male death.
Honchar & Halperin 1981	41.4*		Summary of the 4 USA studies (one unpublished at the time)/ total deaths=105/ three STS cases.

* = Significant at the $p=0.05$ level.

+ = Unquantified excess risk.

Soft Tissue Sarcoma (STS)

Toxicity/Study	Point Estimate of Risk		Comment
	≥1.0	≤1.0	
Case Report Cook 1981			Case report/ one more STS case discovered in the Dow cohort described in Cook, Townsend & Ott.
Moses & Selikoff 1981			Case report/ one more STS case discovered in the Monsanto cohort.
Johnson, Kugler & Brown 1981			Case reports/ two more STS cases with work histories at Monsanto/ not identified as part of above cohorts.
Other Studies Milham 1982		1.2	Proportional Mortality Death certificate study/ 49 STS deaths/ looked at occupations with possible 2,4-D exposure, including farming and forestry.

Reproductive Outcomes

Cohort Studies Smith, Fischer, Pearce & Chapman 1982	548 sprayers in New Zealand and 441 controls. 427 exposed & 352 not exposed one year before or after conception.
Exposed group had 1.19 RR of congenital defect and 0.89 risk of miscarriage. Stillbirths (3) occurred only in the exposed group.	
Townsend et al. 1982	737 conceptions to wives of exposed workers and 2,031 conceptions to wives of unexposed workers at Dow Michigan Division.
No associations demonstrated.	
Other Studies Nelson et al. 1979	Cleft palate occurrence by county with herbicide use by county based on rice acreage.
No association observed.	
Thomas 1980	Congenital malformation and 2,4,5-T use over time in Hungary.
No association observed.	
Hatch 1984	Review of reproductive effects of dioxins/ no definite conclusions.

Neurological Studies

Cohort Studies

Singer, Moses et al.
1982

56 workers in a 2,4-D & 2,4,5-T plant were compared with 25 unexposed controls.

Median motor, median sensory and sural nerve velocities were measured. 46% of exposed group had slowed velocities compared to 5% in the control group ($p < .001$). The mean velocities of the median motor and sural nerves were significantly slower than the controls. There was a highly significant inverse relation between the sural nerve velocity and length of employment. All these relationships remained true when adjusted for alcohol consumption, age and other parameters.

Suskind & Hertzberg
1984

204 workers at Monsanto's Nitro, West Virginia plant exposed to dioxin, and 163 workers not exposed.

Nerve conduction velocities in ulnar and peroneal nerves was non-significantly reduced in exposed group. Sural nerve conduction was slightly reduced in unexposed.

Animal Studies

Sheep and Small Intestinal Adenocarcinoma

Newell, Ross & Renner

Sheep from 88 farms in New Zealand 20,678 female sheep/ 125 cases (6/1000) from 61 farms Exposure to phenoxy herbicides, picolinic herbicide, and combined.

Significant positive trends for treatment of feed with either herbicide or both combined, and with how recently the feed had been treated before consumption.

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Appendix I

Public Participation and Consultation



Appendix I

Public Participation and Consultation

The public was actively involved in the development of the Vegetation Management Draft Environmental Impact Statement. Many people and organizations made valuable contributions to almost every aspect of the analysis.

Setting high goals for active involvement; working with the public early in the process; developing cooperative agreements with state and national agencies; and seeking expert technical and scientific review were some of the actions designed to involve the public in the EIS process.

This appendix describes the roles and activities of the public in more detail.

Issues relating to vegetation management have long been of concern to many people. Over the past years, interaction with the public on vegetation management issues was often controversial and full of conflict. The final result of that era was a court injunction in 1984, prohibiting the use of herbicides until further analysis was completed by the Forest Service.

In developing the new program for vegetation management it was important to “turn over a new leaf” and work to change these previously conflict-charged relationships with the public into collaborative relations.

In keeping with this theme, early meetings were held with key members of the public to identify and develop how to best include the public in the vegetation management decisionmaking that would be taking place. These key players included individuals who surfaced during the 1983-84 litigation as spokespersons for their respective organizations, and who had an interest in working toward mutually

Public Involvement Goals—“Turning Over A New Leaf”

acceptable solutions. They included the Northwest Coalition for Alternatives to Pesticides (NCAP), and Oregonians for Food and Shelter (OFS).

During the course of the initial meetings, a variety of means for involving the public throughout the development of the EIS were worked out. They included:

- periodic mailings of progress reports;
- NCAP and OFS leadership in coordinating involvement of the segments of the public they represent;
- issues workshops for the Forest Service hosted by NCAP and OFS;
- environmental and business community work group assistance to the Forest Service interdisciplinary team;
- a special outreach program to Forest Service employees; and
- close coordination with other interested organizations.

Beginning in June of 1986, many of these actions were implemented. They have served to contact and involve many groups, individuals, and agencies throughout the development of this draft environmental impact statement.

Periodic Mailings: Beginning in June, with the publication of "Request for Participation #1", reports published every two or three months highlighted the status of the EIS, as well as ways people could continue to participate.

These mailing educated both agency employees and the public. They provided a feedback opportunity for information from the public, thus helping to refine and improve the environmental analysis.

Five "Requests for Participation" have been published to date. They will continue to be a important information channel during the comment period and final decisionmaking.

Issue Workshops: As part of their leadership role in coordinating involvement of the they represent, NCAP and OFS conducted issue workshops early in the scoping process. These workshops identified issues these organizations and their representatives felt were critical to the development of the EIS and the vegetation management program.

Working Groups: Throughout the development of the EIS, "working groups" in the environmental, business, and scientific communities provided regular assistance and review to the interdisciplinary (ID) team. They referred technical experts to the ID team; participated in the development of alternatives, and extensively reviewed various portions of the analysis. NCAP and OFS took leader-

ship roles in setting up these “working groups.” Also included were the Western Washington Toxics Coalition (WWTC), and the Washington Pest Management Council (WPMC). Other groups included the Oregon Society of American Foresters.

Employee Outreach: Beginning with “Request for Participation #1”, Forest Service employees have been an integral part of outreach efforts. Forest EIS coordinators provided significant data for analysis; various Forest staff and regional representatives reviewed partial and full drafts of the document; and the expertise of Forest managers was used extensively in specific analyses. The interdisciplinary team also coordinated with other Forest Service Regions, especially in the areas of human health and timber yield analysis.

Other Organizations: Over 100 organizations have been contacted since initial public involvement efforts. Many have participated through NCAP and OFS coordination, and others through Forest Service outreach efforts. Many have made valuable contributions to the EIS and have improved the quality of the document. Continued mailings and other activities during the public comment period are designed to assure that the concerns and perspectives of interested groups and individuals will be heard and considered.

Agencies at all levels of government have a shared interest in resource management. Interagency coordination for development of the new vegetation management program was a specific issue identified during scoping. Many, many agencies have been involved in various aspects of this EIS, and several agencies were designated as formal cooperators.

Cooperating Agencies

Designation of cooperating agencies is a provision of NEPA that emphasizes interagency cooperation early in the EIS process. The lead agency (in this case the Forest Service) may request any other Federal agency with jurisdiction by law or special expertise (with respect to an environmental issue) to be a cooperating agency. A state or local agency of similar qualifications may also become a cooperator through agreement with the lead agency. The cooperating agencies for the development of the vegetation management EIS are:

Environmental Protection Agency, Region X;
Oregon State Department of Environmental Quality;
Oregon State Department of Agriculture;
Oregon State Department of Transportation;
Oregon State University Extension;
Washington State Department of Agriculture;
Washington State Department of Transportation;

Washington State Department of Natural Resources—Forest Lands Division; and

University of Washington—School of Public Health and Community Medicine.

Their environmental analyses, proposals and staff support were used extensively in the development of the EIS.

Review and Analysis

To help the interdisciplinary team improve issue responsiveness, the sharpening of alternatives and development of analysis, a rough (partial) draft of the EIS was made available in March for the cooperating agencies, working groups and internal staff. In response to the comments generated, a second rough draft was prepared for internal review. This second review assured the technical and legal adequacy of the document.

Scientific adequacy is an important part of any environmental analysis. The scientific information behind the DEIS is extensive. Both the University of Washington and Oregon State University have made valuable contributions to the review and analysis presented in the DEIS.

In addition to the Bureau of Land Management/Forest Service, contract with Labat-Anderson, Inc. for a pesticide risk analysis, a contract with the University of Washington, School of Public Health and Community Medicine provided additional expertise in the field of toxicology and public health. Through this arrangement, an independent review of the Labat-Anderson risk analysis was conducted and a systematic evaluation of available information on herbicide toxicity was developed. Members of the University of Washington team included:

Sheldon D. Murphy, Ph.D., Professor & Chair, Department of Environmental Health;

Nicholas Heyer, M.S., (Ph.D. Candidate), Industrial Hygienist;

Gilbert S. Omenn, M.D., Ph.D., Professor and Dean, School of Public Health and Community Medicine;

Lucio G. Costa, Ph.D., Research Associate Professor;

David L. Eaton, Ph.D., Associate Professor; and

Elaine Faustman-Watts, Ph.D., Assistant Professor.

Through cooperation with Oregon State University Extension and the College of Forestry, rough drafts of the DEIS and silviculture appendices were reviewed by Oregon State University specialists. Reviewers included:

John R. Walstad, Ph.D., Professor of Forest Science, Leader of FIR Program;

Steven Radosevich, Ph.D., Professor of Forest Science, Leader of CRAFTS Vegetation Management Research Cooperative;

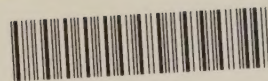
Michael Newton, Ph.D., Professor of Forest Ecology; and

John Tappeiner, Ph.D., Professor of Forest Management.

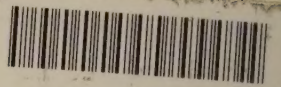
Their comments were considered in preparing the EIS; however, no endorsement of the EIS by Oregon State University or the specialists involved is implied.

Many people have concerns about vegetation management issues. More than concerns, many people feel they have something valuable to contribute. There was a high level of commitment to giving people the opportunity of contributing to the draft analysis. That commitment will continue throughout further development of the Pacific Northwest Region's program for vegetation management.

In Summary



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